E-ISSN: 1309-2014 ISSN: 1309-0720



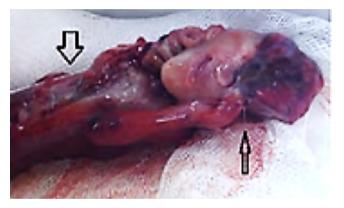
## Clinical and Analytical Medicine

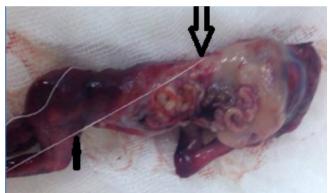
Klinik ve Analitik Tıp Dergisi

Vol: 6 Supplement 5 October 2015

## Amniotic Band Syndrome and a Rarely Seen Subtype: Limb Body Wall Complex

Cırık DA, Altay MM, Kaplan M, Bocutoğlu F, Gelisen O





## Contents;

- · Skin Prick Test Results of Canakkale Onsekizmart University Faculty of Medicine Dermatology Department
- Clinical, Demographic and Laboratory Featuresof Patients with Alopecia Areata in Tokat Region
- Evaluation of Urinary Tract Infections Due to Candida Species
- Treatment of Vaginal Agglutination Complicating Chronic Graft-Versus-Host Disease: A Rare Case Report
- Agammaglobulinemia in a Patient with Smith-Lemli-Opitz Syndrome: Case Report
- Is latrogenic Cushing's a New Form of Child Neglect?
- Co-Existance of Lymph Node Tuberculosis and Pulmonary Embolism: A Case Report
- Importance of CT Imaging on Spontaneous Rupture of Renal Angiomyolipoma: A Case Report
- Methylenetetrahydrofolate Reductase Polymorphisms at Familial Bladder Cancer: Case Report
- Ruptured Ovarian Dermoid Cyst Mimicking Peritoneal Carcinomatosis: CT and MRI
- A Case of Hydatid Cyst with Localization in Anterior Mediastinum
- A Case Report of Telangiectatic Rosacea Treated with Long Pulsed Nd-YAG Laser

E-ISSN: 1309-2014 ISSN: 1309-0720



## Clinical and Analytical Medicine

Klinik ve Analitik Tıp Dergisi

Vol: 6 Supplement 5 October 2015

### DERGI -

Journal of Clinical and Analytical Medicine (Kısa Başlık: J Clin Anal Med) Kartaltepe Mahallesi, Atatürk Bulvarı, Belediye İşhanı, No: 9/9, Bala, Ankara, Türkiye. GSM.:+905303042583 • F.: +90 3128761089 • www.jcam.com.tr • info@jcam.com.tr

Yayıncı	Editör	Sekreter
Derman Tıbbi Yayıncılık	Orhan Yücel	Ülker Bora, Jim Morgan
Kantalana Mila Atatiini Cil Na 0/0	CATA CIIVII C. LI LAD	Kartaltepe Mh, Atatürk Cad.
Kartaltepe Mh. Atatürk Cd. No: 9/9,	GATA Göğüs Cerrahisi. AD.	Kartaitepe Min, Ataturk Cad.
Bala, Ankara, Türkiye.	06118, Etlik, Ankara.	No: 9/9, Bala, Ankara.
T.:+90 3128761089	T.:+905303042583	T.:+90 3123045188
E-posta: info@jcam.com.tr	E-posta: editor@ jcam.com.tr	E-posta: secretary@ jcam.com.tr

Klinik ve Analitik Tıp Dergisi, tıbbın her dalı ile ilgili retrospektif, prospektif veya deneysel çalışmaları, ilginç olgu bildirimlerini, davet üzerine yazılan derlemeleri, editöre mektupları, orijinal görüntüleri, kongre, kurs, seminer, haber ve duyuruları, ilginç araştırmaların özetlerini ve güncel tıp gündemindeki konuları yayınlar. Yayıncılar, reklamı verilen ticari ürünlerin açıklamaları konusunda hiçbir garanti vermemekte ve konuyla ilgili sorumluluk kabul etmemektedir. Dergi yılda 6 sayı olarak Ocak, Mart, Mayıs, Temmuz, Eylül ve Kasım aylarında yayınlanmaktadır. Yazıların bilimsel sorumluluğu yazarlara aittir.

Journal of Clinical and Analytical Medicine publishes every branch of medicine concerned with the retrospective, prospective or experimental studies, interesting case reports, invited reviews, letters to the editor, original images, congress, course, seminar, news item and declaration, brief reports on original studies, and current medical issues in the agenda. Publishers do not give any guarantees about description of the commercial product and do not accept responsibility for the subject. The journal is published six times in a year and in January, March, May, July, September ve November. The author(s) undertake(s) all scientific responsibility for the manuscript.

#### JCAM İndekslendiği Dizinler / Indexs

Embase; Index DOAJ, EMBASE, SCOPUS, Index Copernicus, Pleksus Medline, TÜBİTAK ULAKBİM, Türkiye Atıf Dizini

Matbaa/Yayınevi/Satış/Dağıtım: Derman Tıbbi Yayıncılık, Kartaltepe Mah, Atatürk Cad, No: 9/9, Bala, Ankara, Türkiye. T.: +90 3128761089 • F.: +90 3128761089 • E-Mail: info@jcam.com.tr • Basım Tarihi/Press Data: 01.10.2015

# Clinical and Analytical Medicine

## Editör / Editor

M. Alparslan Turan, Anesthesiology, Anesthesiology Institute, Cleveland Clinic, Cleveland, Ohio, USA Orhan Yücel, Thoracic Surgery, Gülhane Military Medical Academy, Ankara, Turkey

### Bölüm Editörleri / Section Editors

Ali Çelik, Thoracic Surgery, Gazi University Faculty of Medicine, Ankara, Turkey, Ali Sızlan, Anesthesiology, Gulhane Military Medical Academy, Ankara, Turkey, Ayşe Eken, Pharmaceutical Toxicology, Erciyes University, Pharmacy Faculty, Kayseri, Turkey, Betül Battaloğlu İnanç, Family Physician, Mardin Artuklu University, Mardin, Turkey, **Bünyamin Sertoğullarından,** Pulmonology, Van Yüzüncü Yıl University Faculty of Medicine, Van, Turkey, **Didem** Sonbay, Otolaryngology, Antalya Selale Private Medical Center, Antalya, Turkey, Esin Kulaç, Medical Education, Dokuz Eylül University Faculty of Medicine, İzmir, Turkey, Esma Menevşe, Medical Biochemistry, Selçuk University Faculty of Medicine, Konya, Turkey, Hacı Bayram Tosun, Orthopedics and Traumatology, Adıyaman University Faculty of Medicine, Adıyaman, Turkey, Harun Çakmak, Ophthalmology, Adnan Menderes University Faculty of Medicine, Aydın, Turkey, Hasan Senol Çoşkun, Medical Oncology, Akdeniz University Faculty of Medicine, Antalya, Turkey, İlknur Balta, Dermatology, Eskişehir State Hospital, Ankara, Turkey, Kenan Ahmet Türkdoğan, Emergency Medicine, Bezmialem University Faculty of Medicine, İstanbul, Turkey, Mürteza Çakır, Neurosurgery, Atatürk University Faculty of Medicine, Erzurum, Turkey, Mürüvvet Seda Balaban, Hematology, Hacettepe University Faculty of Medicine, Ankara, Turkey, Mustafa Burak Hoşcan, Urology, Medstar Topçular Hospital, Department of Urology, Antalya, Turkey, Nagehan Didem Sarı, Clinical Microbiology and Infectious Diseases, İstanbul TRH, İstanbul, Turkey, Nazif Zeybek, General Surgery, Gulhane Military Medical Academy, Ankara, Turkey, Özgür Pirgon, Pediatric Endocrinology and Diabetes, Süleyman Demirel University, Faculty of Medicine, Isparta, Turkey, Özlem Boybeyi, Pediatric Surgery, Kırıkkale University Faculty of Medicine, Kırıkkale, Turkey, Özlem Erten, Obstetrics and Gynecology, Etlik Zübeyde Hanim Training and Research Hospital, Ankara, Turkey, Şafak Ersöz, Pathology, Karadeniz Technical University Faculty of Medicine, Trabzon, Turkey, Salih Sinan Gültekin, Nuclear Medicine, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey, Vildan Kaya, Radiation Oncology, Süleyman Demirel University School of Medicine, Isparta, Turkey, Yusuf Sinan Şirin, Vet, Surgery, Faculty of Veterinary Medicine, Ankara University, Ankara, Turkey, Zekeriya Arslan, Cardiology, Ankara Mevki Military Hospital, Ankara, Turkey

#### Uluslararası Danışma Kurulu / International Editorial Board,

Abdülkadir Tepeler, Urology, Bezmialem University Faculty of Medicine, İstanbul, Turkey, Abdullah Sivrikaya, Medical Biochemistry, Selçuk University Faculty of Medicine, Konya, Turkey, Abidin Kılınçer, Radiology, Hacettepe University Faculty of Medicine, Ankara, Turkey, Abramson Horacio, Thoracic Surgery, Universidad de Buenos Aires Buenos Aires, Argentina, Abuzer Coşkun, Emergency Medicine, Cumhuriyet University Faculty of Medicine, Sivas, Turkey, Adem Altunkol, Urology, Numune Training and Research hospital, Adana, Turkey, Ahmet Aslan, Orthopedics and Traumatology, Afyonkarahisar State Hospital, Afyonkarahisar, Turkey, Ahmet Gürdal, Cardiology, İstanbul University Faculty of Medicine, İstanbul, Turkey, Ahmet Oğuz Hasdemir, General Surgery, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey, Ahmet Rauf Görür, Thoracic Surgery, GATA Haydarpasa Teaching Hospital, Istanbul, Turkey, Ahmet Şahbaz, Obstetrics and Gynecology, Zonguldak Bülent Ecevit Üniversitesi, Zonguldak, Turkey, Ahmet Şengün, Ophthalmology, Ufuk University Faculty of Medicine, Ankara, Turkey, Ahmet Tolgay Akıncı, Neurosurgery, Trakya University Medical Faculty, Edirne, Turkey, Ahmet Tunç Özdemir, Urology, Yeditepe University Faculty of Medicine, İstanbul, Turkey, Ahmet Uysal, Obstetrics and Gynecology, Onsekiz Mart University Faculty of Medicine, Çanakkale, Turkey, Ali Arslantaş, Neurosurgery, Osmangazi University, Medical Faculty, Eskisehir, Turkey, Ali Ataş, Pediatric, Harran University Faculty of Medicine, Şanlıurfa, Turkey, Ali Karakuş, Emergency Medicine, Mustafa Kemal University Faculty of Medicine, Hatay, Turkey, Ali Kılıçgün, Thoracic Surgery, Abant İzzet Baysal University Faculty of Medicine, Bolu, Turkey, Ali Serdar Gözen, Urology, SLK-Kliniken Urology Department, Heidelberg University, Heilbronn, Germany, Ali Yavuz Karahan, Physical Medicine and Rehabilitation, Beyhekim State Hospital, Konya, Turkey, Alime Güneş, Ophthalmology, Suleyman Demirel University Faculty of Medicine, Isparta, Turkey, Alper Gözübüyük, Thoracic Surgery, Gülhane Military Medical Academy, Ankara, Turkey, Alper Özorak, Urology, Süleyman Demirel University Faculty of Medicine, Isparta, Turkey, Aslı Tanrıvermiş Sayıt, Radiology, Samsun Gazi State Hospital, Samsun, Turkey, Atilla Karateke, Obstetrics and Gynaecology, Hatay Antakya State Hospital, Hatay, Turkey, Aydın Aytekin, Internal Medicine, Medical Faculty of Hacettepe University, Ankara, Turkey, Aykut Recep Aktaş, Radiology, Medical Faculty, Suleyman Demirel University, Isparta, Turkey, Aylin Fidan Korcum, Radiation Oncology, Akdeniz University Faculty of Medicine, Antalya, Turkey, Aynur Özen, Nuclear Medicine, Bagcılar Training and Research Hospital, Istanbul, Turkey, Ayşe İnci, Infectious Diseases and Clinical Microbiology, İstanbul Kanuni Sultan Süleyman TRH, İstanbul, Turkey, Ayşe Neslin Akkoca, Family Medicine, Faculty of Medicine, University of Mustafa Kemal, Hatay, Turkey, Ayşe Nur Demiral, Radiation Oncology, Faculty of Medicine, University of Dokuz Eylül, İzmir, Turkey, Aziz Gümüş, Pulmonology, Recep Tayyip Erdoğan University Faculty of Medicine, Rize, Turkey, **Basar Sareyyupoglu**, Cardiothoracic Surgery, TAMHSC College of Medicine, Texas, USA, **Bekir Eray Kılınç**, Orthopedics and Traumatology, Iqdir State Hospital, İstanbul, Turkey, Berker Özkan, Thoracic Surgery, İstanbul University Faculty of Medicine, İstanbul, Turkey, Betül Battaloğlu İnanç, Family Physician, Mardin Artuklu University, Mardin, Turkey, Bilgehan Savaşöz, Cardiothoracic Surgery, Gülhane Military Medical Academy, Ankara, Turkey, Bülent Altunoluk, Urology, Sütçü İmam University Faculty of Medicine, Kahramanmaraş, Turkey, Bülent Karaman, Radiology, Gulhane Military Medical School, Ankara, Turkey, Burçin Çelik, Thoracic Surgery, 19 Mayis University Medical School, Samsun, Turkey, Cabir Yüksel, Thoracic Surgery, Ankara University Faculty of Medicine, Ankara, Turkey, Can Acıpayam, Pediatric Hematology and Oncology, Mustafa Kemal University School of Medicine, Hatay, Turkey, Can Kürkçüoğlu, Thoracic Surgery, Harran University Faculty of Medicine, Şanlıurfa, Turkey, Cantürk Tasçı, Pulmonary Medicine, Gulhane Military Medical Academy, Ankara, Turkey, Carla Lamb, Pulmonology, Critical Care Medicine, Lahey Clinic, Burlington, USA, Cem Çelik, Obstetrics and Gynecology, Namik Kemal University, Faculty of Medicine, Tekirdağ, Turkey, Cemil Kavalcı, Emergency Medicine, Başkent University Faculty of Medicine, Ankara, Turkey, Crisan Nicolae, Urology, Clinical Municipal Hospital, Cluj-Napoca, Romania, Elmir Memmedov, Baku Bash Klinik Hospital, Baku, Azerbaijan, Emre Delen, Neurosurgery, Edirne State Hospital, Edirne, Turkey , Emre Özkara, Neurosurgery, Osmangazi University, Medical Faculty, Eskisehir, Turkey, Erdal Yekeler, Thoracic Surgery, Ankara High Specialization Training and Research Hospital, Ankara, Turkey, Ergün Tozkoparan, Pulmonary Medicine, Gulhane Military Medical Academy, Ankara, Turkey, Erkan Ceylan, Pulmonology, Medeniyet UniversityMedical School, İstanbul, Turkey,

Erkan Vuralkan, Otolaryngology, Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey, Ersin Zümrütbaş, Urology, Pamukkale University Faculty of Medicine, Denizli, Turkey, Esin Kulaç, Medical Education, Kocaeli University Faculty of Medicine, Kocaeli, Turkey, Esma Menevşe, Medical Biochemistry, Selçuk University Faculty of Medicine, Konya, Turkey, Farah Aziz Khan, Medical Biochemistry, College of Applied Medical Sciences, Shahjahanpur, India, Fatih Candaş, Thoracic Surgery, GATA Haydarpasa Teaching Hospital, Istanbul, Turkey, Fatih Ozcura, Pulmonology, Dumlupınar University Faculty of Medicine, Kütahya, Turkey, Feyzi Yılmaz, Emergency Medicine, Numune Training and Research hospital, Ankara, Turkey, Figen Türk, Thoracic Surgery, Pamukkale University Faculty of Medicine, Denizli, Turkey, Frank-Martin Haecker, Pediatric Surgery, University Children's Hospital, Basel, Gennaro Musi, Urology, Deputy Director, Division of Urology, European Institute of Oncology, Milano, Italy, Gül Soylu Özler, Otolaryngology, Mustafa Kemal University Faculty of Medicine, Hatay, Turkey, Gülay Ok, Anesthesiology, Celal Bayar University Faculty of Medicine, Manisa, Turkey, Gürhan Öz, Thoracic Surgery, Afyon Kocatepe University Faculty of Medicine, Afyon, Turkey, Hacı Bayram Tosun, Orthopedics and Traumatology, Adıyaman University Faculty of Medicine, Adiyaman, Turkey, Hakan Levent Gül, Neurology, Kartal Training and Research Hospital, İstanbul, Turkey, Halit Çınarka, Pulmonology, Recep Tayyip Erdoğan University Faculty of Medicine, Rize, Turkey, Hamza Sucuoğlu, Physical Medicine and Rehabilitation, Silopi State Hospital, Şırnak, Turkey, Hans K. Pilegaard, Cardiothoracic Surgery, Aarhus University Hospital, Denmark, Harun Çakmak, Ophthalmology, Adnan Menderes University Faculty of Medicine, Aydın, Turkey, Hasan Senol Çoşkun, Medical Oncology, Akdeniz University Faculty of Medicine, Antalya, Turkey, Hatice Eryiğit, Thoracic Surgery, Dr. Lütfü Kırdar Kartal Training and Research Hospital, İstanbul, Turkey, Hatice Kılıç, Pulmonology, Atatürk Traning and Research Hospital, Ankara, Turkey, Hayati Bilgic, Pulmonary Medicine, Gulhane Military Medical Academy, Ankara, Turkey, Hülya Aslan, Radiology, Başkent University Faculty of Medicine, Adana, Turkey, Hyung Joo Park, Thoracic and Cardiovascular Surgery, Korea University Medical Center, Korea, İbrahim Haznedaroğlu, Hematology, Hacettepe University Faculty of Medicine, Ankara, Turkey, İlknur Balta, Dermatology, Eskişehir State Hospital, Ankara, Turkey, Ioan Coman, Urology,"Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca, Romania, İsmail Halili, Ophthalmologist, Tirana Central Military Hospital, Tiran, Albania, İsmail Oğuz Kara, Medical Oncology, Çukurova University Faculty of Medicine, Adana, Turkey, İsmail Oskay Kaya, General Surgery, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey, İsmail Şalk, Radiology, Cumhuriyet University Faculty of Medicine, Sivas, Turkey, Kenan Ahmet Türkdoğan, Emergency Medicine, Bezmialem University Faculty of Medicine, İstanbul, Turkey, Koray Aydoğdu, Thoracic Surgery, Atatürk Training and Research Hospital, Ankara, Turkey, Kürşad Zengin, Urology, Bozok University School of Medicine, Yozgat, Turkey, M. Ahmet Tunçkıran, Urology, Başkent University Faculty of Medicine, Antalya, Turkey, Mahmut Kuntay Kokanalı, Obstetrics and Gynecology, Zekai Tahir Burak Training and Research Hospital, Ankara, Turkey, Mahmut Tokur, Thoracic Surgery, Sütçü İmam University Faculty of Medicine, Kahramanmaraş, Turkey, Makbule Ergin, Thoracic Surgery, Gaziosmanpasa University School of Medicine, Tokat, Turkey, Mehmet Dakak, Thoracic Surgery, TOBB ETÜ Hospital, Ankara, Turkey, Mehmet Fatih Korkmaz, Orthopedics and Traumatology, İnönü University Faculty of Medicine, Malatya, Turkey, Mehmet Gamsızkan, Pathology, Erzurum Mareşal Çakmak Military Hospital, Erzurum, Turkey, Mehmet Nuri Erdem, Orthopaedics and Traumatology, Kolan International Hospital, Istanbul, Turkey, Mehmet Yiğit, Emergency Medicine, Bezmialem University Faculty of Medicine, İstanbul, Turkey, Mekin Sezik, Obstetrics and Gynecology, Suleyman Demirel University Faculty of Medicine, Isparta, Turkey, Melike Ceyhan Balcı Şengül, Psychiatrist, Pamukkale University Faculty of Medicine, Denizli, Turkey, Mesut Pişkin, Urology, Necmettin Erbakan University Faculty of Medicine, Konya, Turkey, Muhammet Reha Çelik, Thoracic Surgery, İnönü University Faculty of Medicine,, Turkey, Muharrem Erol, Thoracic Surgery, Bursa University Faculty of Medicine, Bursa, Turkey, Mürteza Çakır, Neurosurgery, Atatürk University Faculty of Medicine, Erzurum, Turkey, Mustafa Burak Sayhan, Emergency Medicine, Trakya University Faculty of Medicine, Edirne, Turkey, Mustafa Kuzucuoğlu, Thoracic Surgery, Dr. İ. Şevki Atasagun State Hospital, Nevşehir, Turkey, Mustafa Okan, İstanbulluoğlu, Urology, Mevlana University Faculty of Medicine, Konya, Turkey, Mustafa Yıldırım, Medical Oncology, Ministry of Health Batman State Hospital, Batman, Turkey, Nagehan Didem Sarı, Clinical Microbiology and Infectious Diseases, İstanbul TRH, İstanbul, Turkey, Nasuh Utku Doğan, Obstetrics and Gynecology, Akdeniz University Faculty of Medicine, Antalya, Turkey, Nazif Elaldi, Clinical Microbiology and Infectious Diseases, Cumhuriyet University Faculty of Medicine, Sivas, Turkey, Necmettin Tanriöver, Neurosurgery, İstanbul University Faculty of Medicine, İstanbul, Turkey, Nesrin Öcal, Pulmonology, Hakkari Military Hospital, Hakkari, Turkey, Nevzat Can Şener, Urology, Adana Training and Research Hospital, Adana, Turkey, Nilgün Yılmaz Demirci, Pulmonology, Gazi University Faculty of Medicine, Ankara, Turkey, Oğuzhan Okutan, Pulmonology, Gülhane Military Medical Academy, İstanbul, Turkey, Okhan Akdur, Emergency Medicine, Çanakkale Onsekiz Mart University Faculty of Medicine, Çanakkale, Turkey, Oktay Karaköse, General Surgery, Süleyman Demirel University Faculty of Medicine, Isparta, Turkey, Ömer Gökhan Doluoğlu, Urology, Ankara Training and Research Hospital, Ankara, Turkey, Ömer Karadaş, Neurology, Erzincan Military Hospital, Erzincan, Turkey, Ömer Önal, Thoracic Surgery, Erciyes University Faculty of Medicine, Kayseri, Turkey, Onur Işık, Cardiothoracic Surgery, Ege University Faculty of Medicine, İzmir, Turkey, Onur Polat, Ophthalmology, Afyonkarahisar State Hospital, Afyonkarahisar, Turkey, Orhan Akpınar, Clinical Microbiology and Infectious Diseases, Süleyman Demirel University, Faculty of Dentistry, Isparta, Turkey, Osman Başpınar, Pediatric Cardiology, Gaziantep University Faculty of Medicine, Gaziantep, Turkey, , Özcan Hız, Physical Medicine and Rehabilitation, Ordu Medikalpark State Hospital, Ordu, Turkey, Özgür Başal, Orthopedics and Traumatology, Süleyman Demirel University Faculty of Medicine, Isparta, Turkey, Özgür Pirgon, Pediatric Endocrinology and Diabetes, S. Demirel University, Faculty of Medicine, Isparta, Turkey, Özgür Samancılar, Thoracic Surgery, Suat Seren Training and Research Hospital, İzmir, Turkey, Özgür Söğüt, Emergency Medicine, Bezmialem University Faculty of Medicine, İstanbul, Turkey, Özlem Boybeyi, Pediatric Surgery, Kırıkkale University Faculty of Medicine, Kırıkkale, Turkey, Peter Goldstraw, Thoracic Surgery, National Heart and Lung Institute, Imperial College, London, UK, Pınar Bıçakçıoğlu, Thoracic Surgery, Ataturk Training and Research Hospital for Chest Disease and Chest Surgery, Ankara, Turkey, Rasih Yazgan, Thoracic Surgery, Süleyman Demirel University Faculty of Medicine, Isparta, Turkey, Raziye Keskin Kurt, Obstetrics and Gynecology, Mustafa Kemal University Faculty of Medicine, Hatay, Turkey, Richard W. Light, Pulmonary Disease, and Critical Care Med., Vanderbilt University, Tennessee, USA, Şafak Ersöz, Pathology, Karadeniz Technical University Faculty of Medicine, Trabzon, Turkey, Salih Sinan Gültekin, Nuclear Medicine, Dışkapı Yıldırım Beyazıt Training and Research Hospital, Ankara, Turkey, Şamil Günay, Thoracic Surgery, Harran University, Faculty of Medicine, Şanlıurfa, Turkey, Seda Özkan, Emergency Medicine, Erciyes University Faculty of Medicine, Kayseri, Turkey, Sedat Abuşoğlu, Medical Biochemistry, Selçuk University Faculty of Medicine, Konya, Turkey, , Selahattin Bedir, Urology, Gülhane Military Medical Academy, Ankara, Turkey, Selen Doğan, Obstetrics and Gynecology, Akdeniz University, Faculty of Medicine, Antalya, Turkey, Süleyman Deniz, Anesthesiology, GATA Haydarpasha Training Hospital, Istanbul, Turkey, Semra Bilaçeroğlu, Pulmonary Medicine, Izmir Training and Research Hospital for Thoracic Medicine and Surgery, Turkey, Serap Karataş Polat, Dermatology, Afyon Kocatepe University Faculty of Medicine, Afyon, Turkey, Serdar Evman, Thoracic Surgery, Süreyyapaşa Training and Research Hospital, İstanbul, Turkey, Serdar Yanık, Pathology, İskenderun State Hospital, İskenderun, Turkey, Şerife Nur Ulusan, Radiology, Başkent University Faculty of Medicine, Adana, Turkey, , Serkan Altınova, Urology, Ankara Atatürk Training and Research Hospital, Ankara, Turkey, Sertaç Yazıcı, Urology, Hacettepe University School of Medicine, Ankara, Turkey, Servet Kayhan, Pulmonary Medicine, Recep Tayyip Erdoğan University, Rize, Turkey, Şevki Mustafa Demiröz, Thoracic Surgery, Atatürk Traning and Research Hospital, Ankara, Turkey, Seyfettin Gümüş, Pulmonary Medicine, Gulhane Military Medical Academy, Ankara, Turkey, Shahinur Rahman, Thoracic Surgery, Combined Military Hospital, Dhaka, Bangladesh, Sinem Özyurt, Nuclear Medicine, Dr. Sami Ulus Children Diseases Hospital, Ankara, Turkey, Soner Şahin, Neurosurgery, Kocaeli Derince Training and Research Hospital, Kocaeli, Turkey, Songül Çuhadaroğlu, Thoracic Surgery, Yedikule Training and Research Hospital, İstanbul, Turkey, Suat Erus, Thoracic Surgery, Artvin State Hospital, Artvin, Turkey, Şükran Ülger, Radiation Oncology, Gazi University Faculty of Medicine, Ankara, Turkey, , Şule Karabulut Gül, Radiation Oncology, Dr. Lütfi Kırdar Kartal Training and Research Hospital, Istanbul, Turkey, Taylan Oksay, Urology, Süleyman Demirel University Faculty of Medicine, Isparta, Turkey, Tevfik İlker Akçam, Thoracic Surgery, Suat Seren Training and Research Hospital, İzmir, Turkey, Ülkü Yazıcı, Thoracic Surgery, Ataturk Chest Diseases and Chest Surgery Training and Research Hospital, Ankara, Turkey, Vildan Kaya, Radiation Oncology, Süleyman Demirel University School of Medicine, Isparta, Turkey, Yoshiya Toyoda, Cardiothoracic Surgery, University of Pittsburgh Physicians, Pittsburgh, USA, Yusuf Peker, General Surgery, Gulhane Military Medical Academy, Ankara, Turkey, Zekeriya Arslan, Cardiology, Ankara Mevki Military Hospital, Ankara, Turkey



## **İçindekiler** (Table of Contents)

## A. Orjinal Araştırma / Original Research

599-602	Gebelikte PCR ile Klamidya Trachomatis ve Neisseria Gonore Taraması Screening of Chlamydia Trachomatis and Neisseria Gonorrhoeae with PCR During Pregnancy Mine Islimye Taskin, Yeşim Alpay
603-605	Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi Dermatoloji Kliniği Deri Prik Test Sonuçları Skin Prick Test Results of Canakkale Onsekizmart University Faculty of Medicine Dermatology Department Selda Işık, Zerrin Öğretmen, Sevilay Kılıç, Sibel Cevizci
606-608	Tokat Bölgesindeki Alopesi Areatalı Hastaların Klinik, Demografik ve Laboratuvar Özellikleri Clinical, Demographic and Laboratory Featuresof Patients with Alopecia Areata in Tokat Region Havva Yıldız Seçkin, Yalçın Baş, Zennure Takçı, Sercan Sezgin
609-611	Candida Türlerine Bağlı İdrar Yolu Enfeksiyonlarının Değerlendirilmesi Evaluation of Urinary Tract Infections Due to Candida Species Yeşer Karaca Derici, Sevgi Yılmaz Hancı, Arzu Bayram, M. Cem Şirin, Pınar Şamlıoğlu, Neval Ağuş, Nisel Yılmaz
612-616	Isparta İli Okul Çağı Çocuklarında İdrar İyot Atılımı ve İyot Eksikliği Durumu Urine Iodine Excretion and Iodine Deficiency Status in School Age Children at Isparta Province Nagehan Aslan, Hüseyin Kurku, Özgür Pirgon

609-611	Evaluation of Urinary Tract Infections Due to Candida Species Yeşer Karaca Derici, Sevgi Yılmaz Hancı, Arzu Bayram, M. Cem Şirin, Pınar Şamlıoğlu, Neval Ağuş, Nisel Yılmaz
612-616	Isparta İli Okul Çağı Çocuklarında İdrar İyot Atılımı ve İyot Eksikliği Durumu Urine lodine Excretion and lodine Deficiency Status in School Age Children at Isparta Province Nagehan Aslan, Hüseyin Kurku, Özgür Pirgon
B. Olg	gu Sunumu / Case Report
617-619	Otuz Altı Yaşında Bir Kadın Hastada Pulmoner Atrezi ve Ventriküler Septal Defekt Birlikteliği Pulmonary Atresia and Ventricular Septal Defect in a Thirty-Six Year Old Woman İbrahim Koç, Ayşen Dökme, Nesimi Yavuz, Gökhan Bulut
620-622	Kronik Lenfositik Lösemi/Küçük Hücreli Lenfositik Lenfomalı Bir Hastada Prostatın Nadir Bir Antitesi: Duktal Adenokarsinoma Ductal Adenocarcinoma: A Rare Entity of Prostate Gland in a Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma Patient Tumay Ozgur, Murat Mehmet Rifaioğlu, Ahmet Taner Sümbül, Füsun Aydoğan, Nesrin Atcı
623-625	Amniyotik Bant Sendromu ve Nadir Görülen Bir Subtipi: Ekstremite Vücut Duvar Kompleksi Amniotic Band Syndrome and a Rarely Seen Subtype: Limb Body Wall Complex Derya Akdağ Cırık, Mehmet Metin Altay, Metin Kaplan, Füsun Bocutoğlu, Orhan Gelisen
626-628	Kronik Graft-Versus-Host Hastalığında Vajinal Aglütinasyon Tedavisi: Olgu Sunumu Treatment of Vaginal Agglutination Complicating Chronic Graft-Versus-Host Disease: A Rare Case Report Suleyman Engin Akhan, Hande Delier, Ercan Bastu, Cenk Yasa, Funda Gungor Ugurlucan, Sevgi Kalayoglu-Besisik
629-631	Erişkinde Torakal Seviyede SCIWORA: Olgu Sunumu SCIWORA at Thoracic Level in an Adult: A Case Report Seniz Akcay Yalbuzdag, Taciser Kaya, Ilker Sengul, Altinay Goksel Karatepe, Zehra Hilal Adibelli
632-634	Adeno Virüs Enfeksiyonuna Bağlı Gelişen ARDS ve Pulmoner Hipertansiyon: Bir Çocuk Olgusu Acute Respiratory Distress Syndrome and Pulmonary Hypertension Induced By Adenovirus Infection: A Child Case Vildan Güngörer
635-637	Karbepenem Dirençli Acinetobacter Pnömonilerinde Tek Başına veya Kombine Kolistin Tedavisi Başarısının Değerlendirilmesi Evaluation of Alone or Combined Colistin Therapy Success in Patients with Carbapenem-Resistant Acinetobacter Pneumonias Özlem Erçen Diken, Ayhanım Tümtürk, Sertaç Arslan
638-640	Agamaglobulinemi ile Seyreden Smith-Lemli-Opitz Sendromu: Olgu Sunumu Agammaglobulinemia in a Patient with Smith-Lemli-Opitz Syndrome: Case Report H. Haluk Akar, Songül Gökay, Turkan Patiroglu, Deniz Okdemir, Mustafa Kendirci
641-643	Penil Protez Migrasyonuna Bağlı Nadir Bir Akut Barsak Tıkanıklığı Olgusu A Rare Case of Acute Intestinal Obstruction Due to the Migration of Penile Prosthesis Savaş Baba, Osman Yıldırım, Murat Özgür Kılıç, Nermin Damla Hatipoğlu, Cem Emir Güldoğan
644-646	İyatrojenik Cushing Yeni Bir Çocuk İhmali Formu Olabilir Mi? Is latrogenic Cushing's a New Form of Child Neglect? Habip Almiş, Ahmet Köse, İbrahim Hakan Bucak, Eda Mengen, Mehmet Turgut
647-649	Uterin Servikse Sessiz Metastaz Yapmış Meme Kanseri Olgusu Breast Cancer with Silent Metastasis to Uterine Cervix Gokhan Pekindil, Pinar Solmaz Hasdemir, Ayca Tan, Gamze Goksel
650-652	Lenf bezi Tüberkülozu ve Pulmoner Emboli Birlikteliği: Olgu Sunumu Co-Existance of Lymph Node Tuberculosis and Pulmonary Embolism: A Case Report İbrahim Koç, Serdar Doğan, Yusuf Doğan, Ahmet Ulusan
653-655	Pelvis Kırığı ile Birlikte Supra Kondiler İskelet Traksiyonu Çivi Dibinden İdrar Fistülü: Olgu Sunumu Bladder Rupture and Urine Fistula Between-Bladder and Supracondylary Pin Tract After Pelvis Fracture: A Case Report Zekeriya Oztemur, Umut Hatay Gölge, Ömer Pıçakçı, Gündüz Tezeren, Okay Bulut

Renal Anjiyomiyolipomların Spontan Rüptüründe BT Görüntülemenin Önemi: Olgu Sunumu Importance of CT Imaging on Spontaneous Rupture of Renal Angiomyolipoma: A Case Report Fatma Gundogdu, Aylin Okur, Halil İbrahim Serin, Sebahattin Albayrak

Atomoksetin Kullanımına Bağlı Üriner Retansiyon: Bir Olgu Sunumu Urinary Retention Associated with Atomoxetine Use: A Case Report Nilfer Sahin, Hatice Altun Toplum Kökenli Pnömoniyi Taklit Eden Pulmoner Emboli Olgusu Pulmonary Embolism Mimicking Community Acquired Pneumonia: A Case Report Ibrahim Koç, Yusuf Doğan, Onur Çakmak, Gökhan Bulut Pulmoner HipertansiyonluBir Hastada Gelişen Transüda Şilotoraks Transudative Chylothorax in a Patient with Pulmonary Hypertension Sedat Kuleci, Oya Baydar, Efraim Güzel, İsmail Hanta Ailesel Mesane Kanserlerinde Metilentetrahidrofolat Redüktaz Polimorfizmleri: Olgu Sunumu Methylenetetrahydrofolate Reductase Polymorphisms at Familial Bladder Cancer: Case Report Gülay Güleç Ceylan, Cavit Ceylan, Seda Yamak Akut Böbrek Yetmezliği olan bir Hastada Watershed Serebral İnfarkt Watershed Cerebral Infarction in a Patient with Acute Renal Failure Ruya Ozelsancak, Ertugrul Erken, Semih Giray, Ozlem Alkar Polimiyozitli Bir Hastada Alt Ekstremite Cerrahisinde Anestezi Deneyimimiz Our Anesthesia Experience for Lower Extremity Surgery in a Patient with Polymyositis Yeliz Kılıç, Tuğba Aşkın, Tülay Dal, Tünay Kandemir, İdil Kaya Hipertrofik KardiyomiyopatiliBir Hastada Çoklu Miyokardiyal Kas Bandı 677-679 Multiple Myocardial Bridges in a Patient with Hypertrophic Cardiomyopathy Tolga Demir, Mazlum Şahin, Semih Öztürk, Hale Bolgi Demir, Ergun Demirsoy Achromobacter Spp.'nin Etken Olduğu Bir Kalkaneus Osteomyeliti 580-681 A Case Report: Osteomyelitis of Calcaneus Caused by Achromobacter Spp. Tuna Demirdal, Ümmü Sena Sarı, Salih Atakan Nemli Nadir Bir Fantosmi Sebebi: Metastatik Kücük Hücreli Karsinom 582-684 A Rare Cause of Phantosmia: Metastatic Small Cell Carcinoma Nihan Coşkun, Ali Ercan Altınöz, Behçet Coşar Posterior Longitüdinal Ligament Osifikasyonuna Bağlı Servikal Miyelopati 85-688 Cervical Myelopathy Induced by Ossification of Posterior Longitudinal Ligament Hidayet Sari, Hamza Sucuoglu, Sarmad Hussain, Ülkü Akarırmak İşkenceye Bağlı Ayak Bileği Deformitesi: Olgu Sunumu Ankle Deformity Associated with Torture: A Case Report Yunus Güzel, Ali Güleç, Mehmet Elmadağ Sinovyal Kistin Neden Olduğu Kubital Tünel Sendromu: Bir Olgu Sunumu Cubital Tunnel Syndrome Due to Synovial Cyst: A Case Report Zahir Kizilay, Ali Yilmaz, Nusret Ok Salmonella Bakteriyemisi: Kronik Granülomatöz Hastalığa Dikkat! Salmonella Bacteremia: Pay Attention to Chronic Granulomatous Disease Fatma Deniz Aygün, Fatih Aygün, Serdar Nepesov, Halit Çam, Yıldız Camcıoğlu Şiddetli Künt Karın Travmasına Bağlı İzole Pnömoperitonyumun Cerrahi Dışı Yönetimi 698-700 Non-Operative Management of Isolated Pneumoperitoneum Due to Severe Blunt Ábdominal Trauma Murat Özgür Kılıç, Mikdat Bozer, Ahmet Türkan, Yeliz Kılıç Peritoneal Karsinomatozisi Taklit Eden Rüptüre Ovaryan Dermoid Kist: BT ve MRG 701-703 Ruptured Ovarian Dermoid Cyst Mimicking Peritoneal Carcinomatosis: CT and MRI Gurcan Erbay, Merve Ozen, Elif Karadeli Afazi Yalnızca Nörolojik bir Bozukluk Mudur? 704-706 Aphasia, Just a Neurological Disorder? Mehmet Tamer Özdemir, Eray Atalay, Bilge Kağan Tur, Şevki Hakan Eren, Ferhat Omurca, Gül Gürsoy Gecikmiş Travmatik İntraserebral Kanama: Hafif Kafa Travmalı Hastalar Kaç Saat İzlenmeli? 707-709 Delayed Traumatic Intracerebral Hemorrhage: For How Many Hours Should Patients with Mild Head Trauma be Observed? Sevdegul Karadas, Hayriye Gonullu, Ismail Gulsen, Huseyin Baltacıoglu Appendiks Olmadan Sağ Alt Kadranda Ağrı Pain in the Right Lower Quadrant Without Appendix Ihsan Yıldız, Yavuz Savas Koca, Koray Okur, Bekir Sarıcık Tekrarlayan Omuz Çıkıklarında Glenoid Displazi: Bir Olgu Sunumu Glenoid Dysplasia in the Recurrent Shoulder Dislocation: A Case Report Hamza Sucuoğlu, Kubilay Ersin Türkmen, İslam Çalışkan, Nurettin İrem Örnek 712-714 Tiplendirilemeyen Haemophilusİnfluenzae Menenjiti: Olgu Sunumu Non-Type B Haemophilus Influenzae Meningitis: A Case Report Fatma Deniz Aygün, Ada Bulut Sinoplu, Fatma Köksal Çakırlar, Haluk Çokuğraş, Yıldız Camcıoğlu

## C. **Derleme** / Review

718-723 Dressler Sendromu
Dressler Syndrome
Erkan Ceylan

### D. Orjinal Görüntü / Original Image

Metakron Akciğer Karsinomlu Olgu A Case of Metachronous Lung Carcinoma Levent Özdemir, Burcu Özdemir

> Anteriyor Mediastinal Yerleşimli Kist Hidatik Olgusu A Case of Hydatid Cyst with Localization in Anterior Mediastinum Gökhan Perincek, Levent Özdemir, Burcu Özdemir

#### F. Editore Mektup / Letter to Editor







CT/NG in Pregnancy / Gebelikte CT/NG

Mine Islimye Taskin<sup>1</sup>, Yeşim Alpay<sup>2</sup> <sup>1</sup>Department of Obstetrics and Gynecology, <sup>2</sup>Department of Infections Diseases and Clinical Microbiology, Balikesir University Faculty of Medicine, Balikesir, Turkey

#### Özet

Neonatal klamidya ve gonore enfeksiyonlarının morbiditesi ve kötü gebelik sonuçları ile olan ilişkisi nedeniyle, CDC tüm gebe kadınların klamidya trachomatis (CT) için ilk prenatal vizitte taranmasını önermektedir. CDC neisseria gonore (NG) taramasını ise yüksek riskli kadınlarda ve prevelansın yüksek olduğu bölgelerde yaşayanlarda önermektedir. Amerikan Kadın Hastalıkları ve Doğum Uzmanları Birliği (ACOG) taramayı 24 yaş ve altındaki gebeler ile herhangi bir yaşta olup yüksek risk faktörü olan gebe kadınlarda önermektedir. ACOG ayrıca gebeliğin üçüncü trimesterinde gebe kadınların tekrar taranmasını önermektedir. Bu çalışmanın amacı gebe kadınlarda CT ve NG enfeksiyon oranlarını üniyersite hastanemizde belirlemektir. Ek olarak gebe popülasyonumuzda seksüel geçişli enfeksiyon hastalıkları risk faktörleri ile CT/NG arasındaki ilişkiyi araştırmak amaçlanmıştır. Bu tanımlayıcı çalışma Balikesir Üniversitesi hastanesi antenatal kliniğine üçüncü trimesterde başvuran gebeler üzerinde yapılmıştır. Hazır soru formları kullanılarak hastaların demografik verileri ve seksüel davranışları ile ilgili bilgiler elde edilmiştir. Seksüel geçişli enfeksiyonların bulguları açısından klinik ve jinekolojik muayeneleri yapılmıştır. Servikal sürüntü örnekleri toplanmış ve polimeraz zincir reaksiyonu (PCR) ile CT ve NG testi yapılmıştır. Tanımlayıcı istatistikler kullanılmıştır. Çalışma grubunun ortalama yaşı 28,9±5,5 olarak bulunmuştur (min-max 19-39). Ortalama gebelik haftası 33,8±3,1; ilk cinsel ilişki yaşı ise 23,3±4,8'dir. Kullanılan kontraseptif yöntemlerden % 38,1 ile koitus interraptus en yüksek orandadır. Lökore hastaların % 85,7'sinde; disparoni % 23,8'inde vardır. CT/NG tüm hastalarda negatif saptanmıştır. Çalışmamızdaki hasta sayısı sınırlı olsa da bu sonuç, bizim popülasyonumuzda pelvik enflamatuar hastalık risk faktörlerinin olmaması nedeniyle, CT ve NG taramasının yararlı olmayacağı hakkında fikir verebilir.

#### Anahtar Kelimeler

Klamidya Trachomatis; Neisseria Gonore; PCR; Gebelik

#### Abstract

Because of association with adverse pregnancy outcomes and the morbid consequences of neonatal chlamydial and gonococcal infections, Centers for Disease Control and Prevention (CDC) recommends that all pregnant women should be screened for chlamydia trachomatis (CT) at the first prenatal visit. CDC also recommends screening for neisseria gonorrhoeae (NG) in women considered at high risk or who live in an area in which the prevalance is high. American College of Obstetricians and Gynecologists (ACOG) recommend screening in pregnant women 24 years of age and younger, as well as women of any age who have high risk. ACOG also recommend rescreening of pregnant women in their third trimester. The purpose of this study was to determine the rates of CT and NG infections in pregnant women in our university hospital. Additionally, we explored the associations between sexually transmitted infection risk factors and CT/NG in pregnant women. This descriptive study was conducted among 42 pregnant women entering an antenatal clinic in Balikesir, Turkey in their third trimester visit. Structured questionnaires were used to collect demographic and sexual behavioral information; clinical and gynecologic examinations were performed to detect clinical signs of sexually transmitted infections. Servical swabs were collected and used to perform polymerase chain reaction (PCR) to detect CT and NG. Descriptive statistics were performed. For the study group, the median age was 28,9±5,5 years (range, 19-39 years). Gestational age was 33,8±3,1 weeks. Age at first sexual intercourse was 23,3±4,8. The most common contraceptive method was coitus interruptus with the percentage of 38,1%. Vaginal discharge was positive 85,7%; dyspareunia was positive 23,8% of the patients. CT/NG was negative in all specimens. Although our study population has limited number; it can give an opinion about screening for NG and CT may not be useful in our population because the lack of risk factors.

Chlamydia Trachomatis; Neisseria Gonorrhoeae; PCR; Pregnancy

DOI: 10 4328/ICAM 3795 Corresponding Author: Mine Islimye Taskin, Department of Obstetrics and Gynecology, Balikesir University School of Medicine, 10145, Çağış Kampüsü, Balıkesir, Turkey. T.: +90 2666121010/4550 F.: +90 2666121294 E-Mail: minetaskin1302@yahoo.com.tr

Seksüel geçişli hastalıklar (STD) dünya çapında kadınlar ve yenidoğanlar için önemli bir halk sağlığı sorunudur; en fazla görülen hastalıklar arasındadır ve hem gelişmiş hem de gelişmekte olan ülkelerde önemli sağlık harcamalarına neden olur. Dünyada her yıl 333 milyon yeni STD vakası tanı almaktadır. Bunların 89 milyonu CT; 62 milyonu NG olup tüm vakaların yaklaşık yarısını oluşturmaktadır [1]. Amerika Birleşik Devletleri verilerine göre CT yıllık 100.000 gebe kadını; NG ise yıllık 13.200 kadını etkilemektedir [2]. Türkiye'de yeterli veri olmamakla birlikte bir çalışmada serolojik yöntemle bakılan CT pozitifliği oranı %17,1 olarak saptanmıştır [3]. Özellikle klamidya enfeksiyonlarının büyük kısmı asemptomatiktir. CT ve NG tedavi edilmediği taktirde kötü gebelik sonuçları ile birliktedir. Spontan düşükler, erken doğum, erken membran rüptürü, doğum sonrası uterin enfeksiyon, konjenital enfeksiyonlar, konjenital anomaliler, düsük doğum ağırlığı bu kötü gebelik sonuçları arasındadır [4]. Bu enfeksiyonlar doğum sırasında anneden bebeğe geçerek oftalmia neonatarum, klamidya pnömonisi, dissemine gonokokal enfeksiyona neden olabilirler [5]. Aktif tedavi edilmemiş CT enfeksiyonu olan gebelerden doğan yenidoğanların %30-50'sinde konjonktivit; %10-20'sinde pnömoni gelişir [6]. Bu enfeksiyonların prenatal tanısı ve tedavisi kötü gebelik sonuçlarının önlenmesinde son derece önemlidir.

Gebe kadınlarda bu enfeksiyon etkenlerini belirlemek için farklı grupların farklı rehberleri vardır. CDC ilk antenatal vizitte gebe kadınların CT açısından taranmasını önermektedir. NG için ise yüksek riskli popülasyonda veya prevelansın yüksek olduğu bölgelerde yaşayanlarda tarama önermektedir. 24 yaş altında olma yüksek risk faktörü olarak değerlendirilmektedir. Erken yaşta cinsel ilişki, multipl seksüel partner, yakın zamanda seksüel partner değiştirmiş olma, düşük sosyoekonomik durum, korunmasız cinsel ilişki, bariyer yöntemini kullanmama diğer risk faktörleridir [7]. CDC, CT pozitif olanlarda tedaviden 3 hafta sonra tekrar kontrol test yapılmasını önermekte; NG için ise tedavi sonrası kontrol teste gerek olmadığını vurgulamaktadır. ACOG, 24 yaşından daha küçük gebe kadınlar ile yüksek riskli grupta tarama önermektedir. ACOG aynı zamanda gebe kadınların üçüncü trimesterde tekrar taranmasını önermektedir [7,8]. Bu bilgiler ışığında, bu çalışmada amacımız CT ve NG pozitifliğini hastanemiz antenatal polikliniğine başvuran gebe kadınlarda araştırmak, risk faktörleri ile ilişkisini belirlemek ve antenatal tarama gerekliliğini değerlendirmektir. Bu çalışma kendi bölgemizde yapılmış ilk çalışma olma niteliğindedir. Türkiye'de gebeler üzerinde bu konuda yapılmış çalışma sayısının yetersiz olması; özellikle NG ile ilgili verilerin az olması nedeniyle çalışma sonuçlarının yararlı olacağını düşünmekteyiz.

#### Gereç ve Yöntem

Çalışmaya 20-40 yaş arası, Ocak 2015 ile Haziran 2015 tarihlerinde Balıkesir Üniversitesi Kadın Hastalıkları ve Doğum polikliniğinde gebe takibine gelmiş ve CT/NG taraması yapılmış, üçüncü trimesterda olan 42 kadın hasta dâhil edilmiştir. CT/NG taraması için örnekler hasta litotomi pozisyonunda iken steril spekulum muayenesi sırasında servikovajinal akıntıdan, steril sürüntü ile alınmış, özel sıvı ortamında laboratuvara ulaştırılmış ve polimeraz zincir reaksiyonu (PCR) yöntemi ile NG ve CT pozitifliği araştırılmıştır. Vajinal sürüntü örnekleri 1 ml sakkaroz fosfat

içeren sıvı medium içine alınmıştır. Daha sonra bu sıvıdan 400 μL alınarak PCR mediumuna eklenmiş ve Xpert CT/NG (Cepheid, Sunnyvale, USA) testini kullanarak CT ve NG araştırılmıştır. Bu real time PCR testinin sensitivitesi ve spesifitesi >%95 üzerindedir. Hastaların demografik verileri, mevcut semptomları, cinsel yolla bulaşan hastalıklar açısından risk faktörleri, ilk cinsel ilişki yaşı, evlilik süresi, partner sayısı, kontraseptif yöntem kullanımı, cinsel ilişki sıklığı, pelvik enflamatuar hastalık öyküsü, cinsel yolla bulaşan hastalık öyküsü sorgulanarak kayıt altına alınmıştır.

#### Bulgular

Hastaların ortalama yaşı 28,9±5,5 olarak bulunmuştur. Başlıca demografik özellikler tablo 1 de verilmiştir. Ortalama gebelik haftası 33,8±3,1; ilk cinsel ilişki yaşı ise 23,3±4,8dir. Gebelerin %38,1'i ilkokul mezunudur. İlk gebeliği olanlar % 61,9 ile en yüksek orandadır. Sadece 1 hastanın yaşam boyu 2 partneri olmuştur. Diğer tüm kadınlar tek eşlidir. Gebelerin üçte birinin gebelikten önce prezervatif kullandığı görülmektedir. Kullanılan kontraseptif yöntemlerden %38,1 ile koitus interaptus en yüksek orandadır. Kondilom öyküsünün 1 hastada olduğu görülmektedir. Lökore hastaların % 85,7'sinde; disparoni % 23,8'inde vardır. CT/ NG tüm hastalarda negatif saptanmıştır (Tablo 2).

Tablo 1. Hastaların demografik özellikleri

Yaş	28,9±5,5
Menarş	13,2±1,3
Evlilik süresi (yıl)	5,7±5,2
Gebelik haftası	33,8±3,1
İlk cinsel ilişki yaşı	23,3±4,8
Eğitim seviyesi	
İlkokul	%38,1
Ortaokul	%21,4
Lise	%14,3
Üniversite	%26,2

#### Tartışma

Bu çalışmanın sonuçları Türkiyede kendi bölgemizde gebelerde CT/NG enfeksiyon önleme ve kontrol stratejileri geliştirmemiz açısından fikir verici niteliktedir. Sonuçlarımıza göre CT/NG enfeksiyonu için üçüncü trimesterde 42 gebe üzerinde PCR gibi yüksek etkinliğe sahip bir yöntemle değerlendirme yapmamıza rağmen pozitiflik saptanmamıştır. Çalışma grubumuzun tek eşli olması, RİA kullanımının düşük olması, erken yaşta koitus oranlarının az olması gibi nedenlerle enfeksiyon oranlarının düşük olduğunu düşünmekteyiz.

Literatürde gebelerde CT/NG prevelansı ile ilgili yapılan çalışmalarda farklı oranlar bulunmaktadır. Peuchant ve ark. [9] yaptığı bir çalışmada CT, NG oranları sırasıyla %2,5 ve %0 olarak saptanmıştır. Bizim çalışmamızda da ise hem CT hem de NG %0 olarak saptanmıştır. Peuchant ve ark. 18-24 yaş gebeler ile partner sayısı 5 ve üzerinde olanlarda CT pozitifliğinin arttığını göstermiştir. Bizim çalışmamızda hastaların tamamına yakınının tek eşli olması, rahim içi araç kullanımının düşük olması gibi nedenlerle CT/NG enfeksiyonu negatif saptanmış olabilir. Ghope ve ark. [10] yayınladığı bir poster sunumunda gebe adölesan

Tablo 2. Hastaların seksüel davranış özellikleri ve CT/NG

Özellik	n	yüzdesi
Önceki doğumlar		
0	26	61,9
1	8	19
2	6	14,3
3	2	4,8
Partner sayısı		
1	41	97,6
>1	1	2,4
Gebelikten önce prezervatif kullanımı		
Evet	15	35,7
Hayır	27	64,3
Kontraseptif yöntem		
Hormonal	3	7,1
RİA	4	9,5
Prezervatif	8	19
Koitus interaptus	16	38,1
Yöntem kullanmayan	11	26,2
Pelvik enflamatuar hastalık öyküsü		
Evet	0	0
Hayır	42	100
Kondilom öyküsü		
Evet	1	2,4
Hayır	41	97,6
Lökore		
Evet	36	85,7
Hayır	6	14,3
Disparonia		
Evet	10	23,8
Hayır	32	76,2
Gebelikte cinsel ilişki sıklığı		
1/hf	18	42,9
2/hf	14	33,3
3/hf	4	9,5
3/ay	4	9,5
1/ay	1	2,4
Cinsel aktif değil	1	2,4
CT/NG		
Pozitif	0	0
Negatif	42	100

grupta CT %0,43; NG %4,31 olarak belirlenmiştir. Chen ve ark. [11] 504 gebe kadın üzerinde yaptıkları bir çalışmada CT %10,1; NG %0,8 olarak saptanmıştır ve bu grup tarama programlarının kendi popülasyonları için uygun olacağını vurgulamışlardır. Çalışma sonuçlarımız göstermiştir ki; hastaların %85'inde lökore pozitif olmasına rağmen CT ve NG tüm hastalarda (n=42) negatif bulunmuştur. Bu bulgu Vuylsteke ve ark. [12] yaptığı çalışmanın bulguları ile uyumludur. Bu çalışmada servikal enfeksiyon ile vajinal akıntı ve karın ağrısı gibi semptomların ilişkili olmadığı gösterilmiştir.

Ülkemizden, 328 cinsel aktif hastanın değerlendirildiği bir çalışmada; vajinal ve endoservikal örnekler, direkt mikroskobi, gram boyama ve kültürler ile değerlendirilmiş ve %1.82 oranında CT, % 0.06 oranında NG saptanmıştır [13]. Yine ülkemizden bir çalışmada erozyone kronik servisiti olan 50 bayan hastanın en-

doservikal örneklerde monoklonal antikor immunokromatografi yöntemiyle CT antijeni değerlendirilmiş; pozitiflik oranı %64 saptanmıştır. Rutin kontrol amacıyla başvuran 23 kadında ise bu oran %21 olarak tespit edilmiştir [14]. Ancak antikor cevabının her zaman akut enfeksiyonu göstermeyebileceği unutulmamalıdır. Ankara'da 146 seks çalışanı kadının örnekleriyle çalışılan bir araştırmada; gonokok ve sifiliz enfeksiyonu tespit edilmemis, CT ve trikomonas vajinalis sıklığı ise sırasıyla %1.4 ve %0.7 olarak saptanmıştır [15]. Yüksek risk grubunda bile görülen bu düsük oranlar; bizim çalışmamızda risk faktörü oldukça az olan gebelerde neden CT ve NG tespit edemediğimizi açıklamaktadır.

Noyan ve ark. nın [3] gebeler üzerinde yaptığı bir çalışmada anti-klamidya IgM antikorları tüm gebelerin %17.1'inde pozitif bulunurken, trimesterlere göre incelendiğinde, IgM antikor prevalansı 1. trimesterde %20 ile en yüksek, 3. trimesterde %11.5 ile en düşük bulunmasına karşın, her üç trimesterdeki değerler arasında istatistiki anlamlı fark saptanmamıştır. Kaldı ki antiklamidya Ig M pozitifliğinin 1-2 aydan 18-24 aya kadar uzayabileceği düşünüldüğünde bu oranların yüksekliği akut enfeksiyonu tamamıyla yansıtmayabilir. Akut enfeksiyonun kesin tanısı için servikal kültür ile etkeni identifiye etmek ya da PCR ile CT genetik materyalinin incelenmesi gerekir. CT zorunlu hücre içi patojen olduğundan standart yayma kültürlerle tespit edilmesi zaten zordur.

Hem CT hem de NG; pelvik enflamatuar hastalık, kronik endometrit, tubal oklüzyon, adezyon gibi nedenlerle infertiliteye yol açabilmektedir. Ülkemizde 90 infertil kadının değerlendirildiği bir çalışmada; klamidya antijen pozitifliği %30 olarak bulunmuştur [16]. Yardımcı üreme teknikleri ile gebelik oluşan 49 olgunun gebelik prognozu incelendiğinde ise CT pozitif olanlarda terme ulaşan gebelik oranı %54.5 iken CT negatif kontrol grubunda bu oran %92.9 olup, istatistiksel olarak anlamlı bulunmuştur [17]. Bizim popülasyonumuz fertil grup olduğundan CT/ NG saptanmaması literatürle uyumlu gözükmektedir. Bir çalışmada maternal enfeksiyon prevelansının %6'nın altında olduğu popülasyonlarda CT taramasının komplikasyonları önlemede açısından faydasının yeterli olmayacağı ve rutin taramanın bu popülasyonda efektif olmayacağı öne sürülmüştür [18].

Urogenital CT/NG tanısında özellikle CT'nin hücre içi bir mikroorganizma olması ve kültürünün zor olması nedeniyle farklı testler tercih edilmektedir. Bu testler nükleik asit amplifikasyon testleri (NAAT), direkt immünfloresans, enzim immünassay ve nükleik asit hibridizasyon testleri olarak sayılabilir. PCR' ın da içinde bulunduğu NAAT en sensitif ve spesifik testlerdir ve tanı ve taramada standart metot haline gelmiştir [7]. Bu çalışmada da akut enfeksiyon bulgusunu göstermede en duyarlı yöntemlerden olan PCR yöntemini kullanarak daha kesin kanaatlara ulaşmak amaçlanmıştır. Kullandığımız Cepheid GeneXpert CT/NG yönteminin değerlendirildiği çok merkezli ve geniş vaka serili bir çalışmada hastaların kendi kendilerine aldığı vajinal sürüntü örnekleri de dahil tüm örneklerde yüksek duyarlılık ve özgüllüğe sahip olduğu saptanmıştır. CT için duyarlılık endoservikal, vajinal ve idrar örneklerinde sırasıyla %97.4, %98.7, %97.6; NG için ise sırasıyla %100, %100 ve %95.6 olarak belirlenmiştir [19].

Çalışmamızın bazı limitasyonları mevcuttur. Bunlardan en önemlisi çalışmadaki denek sayısının sınırlı olmasıdır. Bu ne-

denle çalışmanın sonuçları tarama gerekliliği açısından fikir verici niteliktedir. Buna ilave olarak CT/NG tüm hastalarda negatif saptanmıştır. Çalışma grubumuzda risk faktörlerinin azlığı incelediğimiz enfeksiyon etkenlerinin çok daha nadir görülmesi ile sonuçlanabilir. Bu CT/NG için düşük pozitiflik oranları çok daha geniş hasta grupları ile belirlenebilecektir. Öte yandan, Türkiye'de konu ile ilgili çalışma sayısının sınırlı olması, özellikle CT ve NG'nin birlikte değerlendirildiği çalışma sayısının çok az olması ve PCR gibi etkin bir yöntemin kullanılması bu çalışmanın güçlü yönlerini oluşturmaktadır.

Özetle; gebelik, hormonal değişiklikler ve immünsupresyon nedeniyle CT/NG enfeksiyon riskini artırabilir. Gebelerde bu enfeksiyonların taranması, kadınların çoğu enfeksiyonu asemptomatik geçirdiği ve enfeksiyon tedavi edilmezse uzun süre sebat ettiğinden kötü gebelik sonuçlarının önlenmesinde yararlı olabilir. Ancak bizim toplumumuz için CDC'nin önerdiği tüm gebelere tarama yapmak veya ACOG'un önerdiği gibi gebeleri üçüncü trimesterda tekrar taramak çok uygun gözükmemektedir. Olgu sayımız sınırlı olduğu için çalışmamızdaki veriler fikir verici özelliktedir. Daha büyük hasta grupları ile yapılacak çalışmalar ile bu konuda bir kanaat oluşturmak mümkün olabilecektir.

### Cıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı bevan edilmemiştir.

#### Kaynaklar

- 1. Gerbase AC, Rowley IT, Mertens TE, Global epidemiology of sexually transmitted diseases. Lancet 1998:351(3):2-4
- 2. Blatt AJ, Lieberman JM, Hoover DR, Kaufman HW. Chlamydial and gonococcal testing during pregnancy in the United States. Am J Obstet Gynecol 2012:207(1):55.e1-8.
- 3. Noyan V, Apan TZ, Yücel A, Sağsöz N. Gebe kadınlarda chlamydia trachomatis infeksiyonunun serolojik yöntemlerle taranması. Ankara Üniversitesi Tıp Derg
- 4. Berggren EK, Patchen L. Prevalence of Chlamydia trachomatis and Neisseria gonorrhoeae and repeat infection among pregnant urban adolescents. Sex Transm Dis 2011:38(3):172-4.
- 5 Centers for Disease Control and Prevention, sexually transmitted diseases treatment guidelines 2006. MMWR Morb Mortal Wkly Rep 2006;55;1-94
- 6. Bébéar C, de Barbeyrac B. Genital Chlamydia trachomatis infections. Clin Microbiol Infect 2009;15:4-10.
- 7. Keegan MB, Diedrich JT, Peipert JF. Chlamydia trachomatis infection: Screening and Management. J Clin Outcomes Manag 2014;21(1):30-8.
- 8. Centers for Disease Control and Prevention. Sexually transmitted diseases treatment guidelines 2010. MMWR Morb Mortal Wkly Rep 2010;59;1-110.
- 9. Peuchant O. Le Roy C. Desveaux C. Paris A. Asselineau I. Maldonado C. Chêne G. Horovitz J, Dallay D, de Barbeyrac B, Bébéar C. Screening for Chlamydia trachomatis, Neisseria gonorrhoeae, and Mycoplasma genitalium should it be integrated into routine pregnancy care in French young pregnant women? Diagn Microbiol Infect Dis 2015;82(1):14-9.
- 10. Ghope P, Sonkar SC, Wasnik K, Mittal P, Saluja D. Prevalence of Chlamydia trachomatis, Neisseria gonorrhoeae and Trichomonas vaginalis infection in pregnant adolescent women and its association with pregnancy outcomes. BMC Infectious Diseases 2014:14(3):E33
- 11. Chen XS, Yin YP, Chen LP, Thuy NT, Zhang GY, Shi MQ, Hu LH, Yu YH. Sexually transmitted infections among pregnant women attending an antenatal clinic in Fuzhou, China, Sex Transm Dis 2006;33(5):296-301.
- 12. Vuylsteke B, Laga M, Alary M, Gerniers MM, Lebughe JP, Nzila N, Behets F, Van Dyck E, Piot P. Clinical algorithms for the screening of women for gonococcal and chlamydial infection: evaluation of pregnant women and prostitutes in Zaire. Clin Infect Dis 1993;17(1):82-8.
- 13. Baksu B, Baksu A, Çınar S, Ağar E, Davas İ, Akvardar T. Vajinal Akıntıda Mikrobiyolojik Değerlendirme. Jinekoloji ve Obstetrik Derg 2005;19(1):50-2.
- 14. Açar A, Sipahi AB, Onan MA, Kubatova A, Sultan N. Kronik erozyone servisitli hastalarda Chlamydia trachomatis antijen sıklığının araştırılması. Türk Jinekoloji ve Obstetrik Derneği Derg 2008;5(1):57-61.
- 15. Zarakolu P. Alp S. Yağcı S. Ankara İlinde Kayıtlı Seks Calısanı Kadınlarda İvilestirilebilir Cinsel Yolla Bulaşan Enfeksiyonların Sıklığı. Mikrobiyol Bul 2010;44:117-21
- 16. Sırmatel F, Telli E, Bayrak S, Sırmatel Ö. İnfertilite Araştırmalarında Chlamydia Trachomatis. Ankara Üniversitesi Tıp Derg 1999;52(4):227-9.

- 17. Göker Taymergen E. Özcakır HT. Levi R. Adakan S. Taymergen E. ART uygulamalarında clamidya enfeksiyonunun gebelik oranları ve prognozuna olan etkisi. Gynecology Obstetrics and Reproduction Medicine 2000;6(3):192-3.
- 18. Carroll JC. Chlamydia trachomatis during pregnancy. To screen or not to screen? Can Fam Physician 1993;39:97-102.
- 19. Gaydos CA, Van Der Pol B, Jett-Goheen M, Barnes M, Quinn N, Clark C, Daniel GE, Dixon PB, Hook EW 3rd; CT/NG Study Group. Performance of the Cepheid CT/ NG Xpert Rapid PCR Test for Detection of Chlamydia trachomatis and Neisseria gonorrhoeae. J Clin Microbiol 2013;51:1666-72.

#### How to cite this article:

Islimye Taskin M, Alpay Y. Screening of Chlamydia Trachomatis and Neisseria Gonorrhoeae with PCR During Pregnancy. J Clin Anal Med 2015;6(suppl 5): 599-602.

## Skin Prick Test Results of Canakkale Onsekizmart **University Faculty of Medicine Dermatology Department**



Çanakkale Onsekiz Mart Üniversitesi Prick Test Sonuçları / Skin Prick Test Results of Canakkale Onsekizmart University

Selda Işık<sup>1</sup>, Zerrin Öğretmen<sup>1</sup>, Sevilay Kılıç<sup>1</sup>, Sibel Cevizci<sup>2</sup> <sup>1</sup>Dermatoloji AD. <sup>2</sup>Halk Sağlığı AD. Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi, Çanakkale, Türkiye

COMÜ klinik araştırmalar etik kurul başkanlığı onay tarih: 28.2.2013. sayı: 050.99-69 Çalışmamız 13-17 kasım 2013 tarihli XXI. Prof. Dr. A. Lütfü Tat Sempozyumunda poster olarak sunulmuştur.

#### Özet

Amaç: Çalışmamızda Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi Dermatoloji polikliniğine başvuran kronik ürtiker, allerjik rinit, sinüzit, konjunktivit, farenjit, allerjik astma, atopik dermatit tanılı hastaların deri prik test sonuçlarının retrospektif olarak değerlendirilmesi amaçlanmaktadır. Gereç ve Yöntem: Bölümümüzde kronik ürtiker, allerjik rinit, sinüzit, konjunktivit, farenjit, allerjik astma, atopik dermatit tanısı almış ve prik test uygulanmış 583 hastanın test sonuçları retrospektif olarak değerlendirildi. Test içeriğinde polenler, otlar, mantarlar, lateks, kedi-köpek tüyü, ev tozu akarı ve gıdalardan oluşan 50 adet standart allerjen çözelti mevcuttu. Bulgular: Hastaların yaşları 3-70 yaş arasında olup, yaş ortalaması 30,6±17,5'du. 359 hastada (%61,6) herhangi bir allerjene pozitiflik tespit edildi. En sık görülen allerjenler; ev tozu akarları (%50,5), otlar (%28,8), hububat polenleri (%29), gıdalardan ise domates (%13,7), kahve (%13,1), tavuk eti (%13,0) ve çikolata (%12,7) idi. Tartışma: Çanakkale'de elde ettiğimiz deri prik test sonuçlarının ülkemiz allerjen haritasına katkıda bulunacağı düşüncesindeyiz.

#### Anahtar Kelimeler

Prick Test; Atopi; Allerji

#### Abstract

Aim: In this study we aimed to evaluate the prick test results of the patients with chronic urticaria, allergic rhinitis, sinusitis conjunctivitis, pharyngitis, allergic asthma and atopic dermatitis who attended to dermatology department of Canakkale Onsekiz Mart University Faculty of Medicine, Material and Method: The prick test results of 583 patients with chronic urticaria, allergic rhinitis, sinusitis, conjunctivitis, pharyngitis, allergic asthma, atopic dermatitis were assessed retrospectively for this study. 50 standard allergens including pollens, grass, weed, fungal allergens, latex, cat fur, dog hair, house dust mites and foods were performed to the patients. Results: The ages of the patients were between 3 and 70 (mean age was 30.6±17.5). At least one positive prick test result to any allergen were obtained in 359 patients (61.6%). The most common allergens were; house dust mites (50.5%), weed(28.8%), polen grains(29%), tomato (13.7%), coffee (13.1%), chicken meat (13.0%) and chocolate (12.7%), respectively. Discussion: We think that our study will help to contribute to the allergen map of our country.

#### Keywords

Prick Test; Atopy; Allergy

Received: 06.07.2015 Accepted: 16.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 603-5 DOI: 10.4328/ICAM.3746 Corresponding Author: Sevilay Kılıç, Dermatoloji A.D, Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi, 17100, Kepez, Çanakkale, Türkiye. GSM: +905377930419 F.: +90 2862635957 E-Mail: sevilay.oguz@gmail.com

IgE'ye bağlı hipersensivite reaksiyonlarının tanısında allerji testlerinin önemli bir yeri vardır. Yüksek etkinlik, düşük maliyet gibi nedenlerle deri prik testleri, aeroallerjenler, gıdalar, ilaçlar gibi bazı antijenlere karşı tip 1 aşırı duyarlılık oluşturan (allerjik rinit, allerjik konjunktivit, allerjik astma, atopik dermatit gibi) hastalıkların tanısında halen yüksek etkinlik, düşük maliyet gibi nedenlerle birincil test olarak tercih edilmektedir [1-3]. Çalışmamızda atopi öyküsü olan 583 hastanın prik test sonuçlarını retrospektif olarak inceleyerek, bölgemizde ağırlıklı olarak rol alan allerjenleri belirlemeyi amaçladık.

#### Gereç ve Yöntem

Çalışmaya Çanakkale Onsekiz Mart Üniversitesi (ÇOMÜ) klinik araştırmalar etik kurul başkanlığından onay alındıktan sonra ÇOMÜ Tıp Fakültesi Dermatoloji polikliniğine başvuran kronik ürtiker, allerjik rinit, sinüzit, konjunktivit, farenjit, allerjik astma, atopik dermatit tanısı olan ve prik test uygulanmış 583 hastanın test sonuçları dahil edildi. Tüm hastaların detaylı anamnez ve fizik muayene bilgileri değerlendirildi. Prik test uygulanmadan önceki 15 gün içerisinde antihistaminik, kortikosteroid, bronkodilatatör, mast hücre stabilizatörleri, immünsüpresif ajanlar gibi test sonucunu etkileyebilecek topikal veya sistemik ilaç kullanan hastalar çalışmaya dahil edilmedi. Test için polenler, otlar, yabani otlar, mantarlar, lateks, epitel, tüy, ev tozu akarı ve gıdalardan oluşan 50 adet standart aktivite ve konsantrasyondaki allerjen çözeltiler kullanıldı (Stallergenes®, Albio®). Allerjenler ön kolun iç yüzüne, ucu 1 mm olan tek kullanımlık lansetler yardımıyla epidermise sızdırılarak uygulandı. Ciltte oluşan reaksiyon 20 dakika sonra değerlendirildi. Deride oluşan eritem ve ödem negatif (temoin) ve pozitif (histamin hidroklorit 1mg/ml) kontrolle karşılaştırıldı. Pozitif kontrolün oluşturduğu ürtikeryal papülün yarısı veya daha büyük çapta papül oluşturan allerjen pozitif kabul edildi.

Veri girişi ve analizi için SPSS istatistiksel 19,0 paket programı kullanıldı. Tanımlayıcı verilerin sunumunda sayı, yüzde, ortalama ve standart sapma değerleri; gruplar arası karşılaştırmalarda kategorik değişkenler için Fisher's kesin testi kullanıldı. p<0.05 değeri "istatistiksel olarak anlamlı" kabul edildi.

### Bulgular

Hastaların yaşları 3-70 yaş arasında olup, yaş ortalaması 30,6±17,5'du. Hastaların 207'si (%35,5) erkek, 376'i (%64,5) kadındı. 309 hastada bakılan IgE ortalaması ise 209,3±681,1'di. Çalışma grubumuzda hastaların (n=583), hastalık gruplarına göre dağılımı incelendiğinde %0.5'inde (n=3) anjiyoödem, %4.8'inde (n=28) astım, %0.5'inde (n=3) astım ve ürtiker, %1.4'ünde (n=8) atopik dermatit, %0.5'inde (n=3) farenjit, %2.1'inde (n=12) pruritus, %25,9'unda (n=151) rinit, %0.2'sinde (n=1) rinit ve astım, %0.2'sinde (n=1) rinit ve konjuktivit, %61.9'unda (n=361) ürtiker, %2'sinde (n=12) ürtiker ve rinit saptandı.

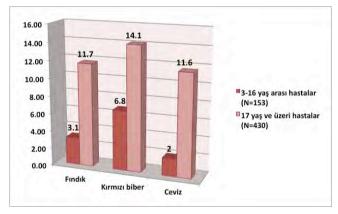
Tüm hastalıklarda en sık görülen allerjenler; ev tozu akarları %50,5 (dermatophagoides pteronyssinus %25, dermatophagoides farinae %25,5), otlar %28,8, hububat polenleri %29, yulaf, buğday, arpa, mısır'dan oluşan dört hububat karışımı % 18,5, arpa, mısır, pirinç, çavdar, buğday, yulaf, buğday unundan oluşan yedi hububat karışımı %10,5, kedi tüyü %14,7, köpek tüyü %16,3, polenler %9,5, küf mantarları %9,1 ve zeytin ağacı %8,8, gıdalardan ise domates %13,7, çikolata %12,7, tavuk

eti %13,0, kahve %13,1, karabiber %12,2, kırmızıbiber %12,2, limon %12,3, mısır %11,8, yumurta akı %11,8, çilek %11,5, köri %11,1, fındık %9,8, ceviz %9,1 idi.

Çalışma grubumuzda allerjenlerin dağılımı hastalık tanısı alt gruplarında da incelendi. Ürtiker tanısı alanların %65.1'inde (n=235) prick testi pozitifti. Bu hastalarda en sık görülen allerjenler sırasıyla %27.5 (n=73) evtozuiki, %26.4 (n=70) evtozubir, %22.6 (n=60) oniki ot, %20.0 (n=53) Dört hububat, %19.7 (n=52) köpek tüyü ve %16.3 (n=43) kedi tüyüydü. Rinit tanısı alanların %53.6'sında (n=81) prick testi pozitifti. Bu hastalarda en sık görülen allerjenler sırasıyla %21.4 (n=27) oniki ot, %19.0 (n=24) Dört hububat, %18.3 (n=23) evtozubir, %16.7 (n=21) evtozuiki, %11.1 (n=14) kedi tüyü ve %7.9 (n=10) köpek tüyüydü. Astım tanısı alanların %53.6'sında (n=15) prick testi pozitifti. Bu hastalarda en sık görülen allerjenler sırasıyla %40.0 (n=10) evtozubir, %40.0 (n=10) evtozuiki, %24.0 (n=6) oniki ot, %8.0 (n=2) Dört hububat, %12.0 (n=3) köpek tüyü ve %8.0 (n=2) kedi tüyüydü. Pruritus tanısı alan 7 kişinin prick testi sonucu pozitifti. Bu hastalarda görülen allerjenler evtozubir (n=3), evtozuiki (n=2), köpek tüyü (n=2), yumurta akı (n=1), kahve (n=1), oniki ot (n=1), dört hububat (n=1), karabiber (n=1), inek sütü (n=1) ve vaban arasıydı (n=1).

Çalışma grubumuzda cinsiyete göre prick test pozitifliği açısından istatistiksel anlamlı bir fark bulunmadı (p=0.924).

Çalışma grubumuzda 3-16 yaş grubunda 153 hasta (%26,2), 17 yaş ve üzeri grupta 430 hasta (%73,8) vardı. Onyedi yaş ve üzeri hasta grubunda fındık (n=28), kırmızı biber (n=46) ve ceviz allerjisi (n=17), 3-16 yaş grubuna göre (sırasıyla n=2, n=8, n=1) istatistiksel olarak anlamlı düzeyde daha yüksekti (p<0,05) (Grafik 1). Diğer allerjenler açısından yaş grupları arasında ista-



Grafik 1. Çalışma grubumuzda fındık, kırmızı biber ve ceviz alerjenlerinin pozitiflik durumu, Çanakkale, 2014

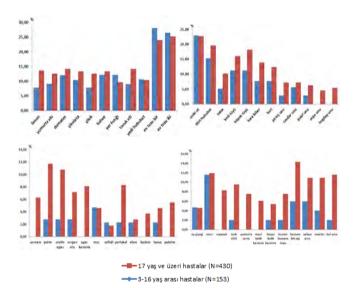
tistiksel olarak bir fark bulunmadı (p>0.05). Çalışma grubumuzda yer alan hastalarda yaş gruplarına göre prick testi sonucuna göre allerjenlerin dağılımları Tablo 1'de ve grafiklerle sunulmuştur (Grafik 2).

#### **Tartısma**

Kliniğimizde uygulanan prik testlerde %61,6 pozitiflik saptandı. Ülkemizde yapılan benzer çalışmalarda farklı oranlar bildirildiğini gözlemledik. İzmir Atatürk Eğitim ve araştırma hastanesinde atopi, atopik dermatit, kronik ürtiker, allerjik rinit, sinüzit ve konjunktivitli 550 hastada yapılan çalışmada %44,36 pozitiflik bildirilmektedir [4]. Lokaj-Berisha ve ark. [5] allerjik astma ve rinitli hastalarda %81,3, Öztürk ve ark. [6] allerjik rinitli 180 has-

Tablo 1. Çalışma grubumuzda yaş gruplarına göre allerjenlerin dağılımı, Çanakkale, 2013

Hasta grupları		Allerjenler (%)										
	limon	yumurta akı	domates	çikolata	çilek	kahve	yer fıstığı	tavuk eti	yedi hububat	ev tozu bir	ev tozu iki	
3-16 yaş arası hastalar (N=153)	7,70	9,10	11,90	10,40	7,70	12,10	12,10	9,00	10,60	28,00	26,30	
17 yaş ve üzeri hastalar (N=430)	13,60	12,50	14,20	13,30	12,50	13,30	9,60	14,20	10,40	23,90	25,20	
	oniki ot	dört hububat	latex	kedi tüyü	köpek tüyü	kara biber	kori	pirinç unu	cavdar unu	yulaf unu	arpa unu	bugday unu
3-16 yaş arası hastalar (N=153)	22,90	15,30	5,10	11,10	11,10	7,70	7,70	2,80	5,60	2,80	0,00	0,00
17 yaş ve üzeri hastalar (N=430)	22,70	19,60	10,10	16,00	18,10	13,80	12,30	7,20	7,20	6,30	4,50	5,40
	sarıcam	polen	zeytin agacı	ısırgan otu	agac karısımı	muz	seftali	portakal	elma	badem	havuç	patates
3-16 yaş arası hastalar (N=153)	0,00	2,80	2,80	2,80	0,00	4,70	2,30	2,30	2,30	0,00	2,30	0,00
17 yaş ve üzeri hastalar (N=430)	6,30	11,70	10,80	7,20	8,10	4,60	1,80	8,30	2,80	3,70	4,60	5,50
	ay çiçegi	mısır	ıspanak	inek sütü	yumurta sarısı	mavi balık karısımı	beyaz balık karısımı	kumes hayvanı tuyu	hamam böcegi	yaban arısı	mantar	bal arısı
3-16 yaş arası hastalar (N=153)	4,70	11,60	0,00	2,00	0,00	0,00	2,00	2,00	6,00	6,00	4,00	2,00
17 yaş ve üzeri hastalar (N=430)	4,60	11,90	8,30	9,50	7,50	6,10	5,40	7,50	14,30	10,90	10,90	11,60



Grafik 2. Çalışma grubumuzda allerjenlerin dağılımı, Çanakkale, 2014

tada %56,7, Akaya ve ark. [7] allerjik astma ve allerjik rinitli hastalarda %57, Tezcan ve ark. [8] atopisi veya ailesel atopi öyküsü olan 5055 hastada %48, Mısırlıoğlu ve ark. [9] 543 alerjik rinitli çocuk hastalarda %24,8, Çalışkaner ve ark. [10] kronik ürtikerli hastalarda %27,4 pozitiflik saptamışlardır. Oranlardaki bu farklılıkların bölgesel allerjenlerin dağılımından ve çeşitliliğinden kaynaklandığını düşünmekteyiz.

Çalışmamızda en yüksek pozitiflik oranınını ev tozu akarlarına karşı tespit ettik (%50,5). Birçok araştırmacı da en yüksek pozitiflik oranını ev tozu akarlarına karşı bildirmiştir. Akaya ve ark. [7] %45, Tezcan ve ark. [8] %42, Çalışkaner ve ark. [10] %24,7, Öztürk ve ark. [6] %72.5 ile %63.7 (sırasıyla; Dermatophagoides pteronyssinus ve Dermatophagoides farinae) ev tozu akarlarına pozitiflik bildirirken, İzmir'de [4] bu oran %11,63 olarak saptanmıştır. Çanakkale'de akarlara karşı olan yüksek pozitivitenin yüksek nem oranına ve rüzgarla taşınan farklı toz partiküllerinin yoğun olmasına bağlı olduğunu düşünmekteyiz. Literatürde de bu durumu destekler nitelikte ev tozlarında ve gıdalarda bulunan akarların bölgesel çalışmalarla ortaya konması gerektiği bildirilmiştir [11,12].

Akarlardan sonra en yüksek oranda otlar (%22,9), hububat polenleri (%18,7), kedi tüyü (%14,7) ve köpek tüyü (%16,1) polenler (%10,1), küf mantarları (%10,1) ve zeytin ağacına (%9,5) karşı duyarlılık tespit ettik. Özellikle zeytin ağacının yaygın bulunduğu bir yörede bulunmamız çevresel faktörlerin, doğal bitki örtüsünün etyolojide rol aldığını destekler niteliktedir.

Gıdalardan en yüksek oranda domates'e (%13,5) karşı duyarlılı-

ğın gözlenmesi de yörede domates üretiminin yaygın olmasına bağlı olarak bölgesel farklılıkların etkisini göstermektedir.

Gıda allerjileri daha çok çocukluk döneminde görülürken, erişkin yaşlarda azalmaktadır. Çalışmamızda ise bu farklılık tespit edilmemiştir. Bunun araştırmamızda çocuk hasta sayısının az olmasına bağlı olabileceğini düşünmekteyiz.

Sonuç olarak; tip 1 aşırı duyarlılığa bağlı hastalıklarda en sık kullanılan test olan prik testin kullanımıyla, allerjenlerin tespit edilmesinde ve bireylerin yaşam kalitesinin artırılmasında önemli adımlar atılmıştır. Çanakkale'de elde ettiğimiz sonuçların da bölgemiz ve ülkemiz allerjen haritasına katkıda bulunacağı düşüncesindeyiz.

#### Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı beyan edilmemiştir.

#### Kavnaklar

- 1. Van Kampen V, de Blay F, Folletti I, Kobierski P, Moscato G, Olivieri M, et al. Evaluation of commercial skin prick test solutions for selected occupational allergens. Allergy 2013;68(5):651-8.
- 2. Bernstein IL, Li JT, Bernstein DI, Hamilton R, Spector SL, Tan R, et al. Allergy diagnostic testing: an updated practice parameter. Ann Allergy Asthma Immunol 2008;100 (3 Suppl 3):1-148.
- 3. Bains P, Dogra A. Skin prick test in patients with chronic allergic skin disorders. Indian J Dermatol 2015;60(2):159-64.
- 4. Öğretmen Z, Güven F, Aydın O, Bozdağ KE, Bilgin İ, Cengiz Bıçakçı ve ark. İzmir Atatürk Eğitim Ve Araştırma Hastanesi Dermatoloji Kliniği Prick Test Sonuçları. Türkiye Klinikleri J Dermatol 2005;15(3):125-8.
- 5. Lokaj-Berisha V, Berisha N, Lumezi B, Ahmetaj L, Bejtullahu G, Karahoda N et al. Sensitization to aeroallergens in patients with respiratory allergies based on skin-prick test results. Iranian J public Health 2012;41(10):29-35.
- 6. Öztürk Ö, Tokmak A, Güçlü E, Yıldızbaş Ş, Gültekin E. Düzce'de Allerjik Rinitli Hastalarda Prick Testi Sonuçları. Konuralp Tıp Dergisi 2005;1:11-14.
- 7. Akaya A, Ünlü M, Uygun N. Isparta Yöresinde Alerjik Astma ve Alerjik Rinitli Olgularda Prick Test ve Total IgE Sonuçlarının Değerlendirilmesi. SDÜ Tıp Fakültesi Dergisi 1995:2:29-32.
- 8. Tezcan D, Uzuner N, Sule Turgut C, Karaman O, Köse S. Retrospective evaluation of epidermal skin prick tests in patients living in Aegean region. Allergol Immunopathol 2003;31(4):26-30.
- 9. Mısırlıoğlu ED, Cengizlier R. Perennial ve Mevsimsel Alerjik Rinitli Çocukların Değerlendirilmesi. Asthma Allergy Immunol 2003;1:11-6.
- 10. Çalışkaner Z, Öztürk S, Turan M, Karaayvaz M. Skin test positivity to aeroallergens in the patients with chronic urticaria without allergic respiratory disease. J Investig Allergol Clin Immunol 2004;14(1):50-4.
- 11. Cevizci S, Gökçe S, Bostan K, Kaypmaz A. Depo gıdalarını ve peynirleri enfeste eden akarlara halk sağlığı açısından bakış. Turkiye Parazitol Derg 2010;34(3):191-
- 12. Gökçe S, Cevizci S, Kaypmaz A. Halk sağlığı penceresinden ev tozu akarları. TAF Prev Med Bull 2010;9(6):695-702.

Işık S, Öğretmen Z, Kılıç S, Cevizci S. Skin Prick Test Results of Canakkale Onsekizmart University Faculty of Medicine Dermatology Department. J Clin Anal Med 2015;6(suppl 5): 603-5.

## Clinical, Demographic and Laboratory Features of Patients with Alopecia Areata in Tokat Region



Tokat Bölgesindeki Alopesi Areatalı Hastaların Klinik, Demografik ve Laboratuvar Özellikleri

Tokat Bölgesindeki Alopesi Areatalı Hastaların Özellikleri / Features of Patients with Alopecia Areata in Tokat Region

Havva Yıldız Seçkin, Yalçın Baş, Zennure Takçı, Sercan Sezgin Gaziosmanpasa University, School of Medicine, Department of Dermatology, Tokat, Turkey

#### Özet

Amaç: Alopesi areata kıl foliküllerini etkileyerek yama tarzı saç kaybıyla seyreden, yaş ve cinsiyet farkı gözetmeksizin gelişebilen kronik inflamatuar bir hastalıktır. Bu çalışmada bölgemizdeki alopesi areata tanılı hastaların klinik, demografik ve laboratuvar özelliklerinin incelenmesi amaçlanmıştır. Gereç ve Yöntem: Gaziosmanpaşa Üniversitesi Tıp Fakültesi, Deri ve Zührevi Hastalıklar Anabilim Dalı Kliniği'ne Ocak 2013-Ocak 2015 tarihleri arasında başvuran alopesi areata tanısı alan 246 hastanın verileri retrospektif olarak incelendi. Laboratuvar özelliklerinin karşılaştırılması için yaş ve cinsiyet özellikleri benzer olan 87 sağlıklı kontrol çalışmaya dahil edildi. Bulgular: Çalışmaya 101'i kadın (%41,1) ve 145'i erkek (%58,9) toplam 246 hasta alındı. Hastaların yaş ortalaması 29,84±10,92 idi. Hastaların 31'i çocuk (<18 yaş), %89,4'ü 40 yaş altında ve %67,5'i 20-40 yaş arasındaydı. Hastaların 240'ında (%97,5) AA, 5'inde (%2) alopesi totalis, 1'inde (%0,4) alopesi üniversalis mevcuttu. Hastaların %23'ünde hastalığın başlangıç yaşının 20 yaşın altında olduğu saptandı. Saçlı deride hastaların %95,9'unda hafif tutulum ve %4,1'inde şiddetli tutlum vardı. Hastaların 69'unda (%28) tırnak tutulumu tespit edildi. Saclı deride hastalık şiddeti ile cinsiyet, tırnak tutulumu ve hastalık başlangıç dönemi arasında anlamlı bir ilişki bulunmadı (p>0,05). Hastaların 18'inde (%7,3) aile hikayesi mevcuttu. Aile hikayesi ile hastalığın başlangıç dönemi ve hastalık şiddeti arasında anlamlı bir ilişki saptanmadı (p>0,05). Hastaların laboratuar özellikleri kontrollerle karşılaştırıldığında, serbest T3, serbest T4, hemogram, ferritin, vitamin B12 ve folat değerleri açısından istatistiksel olarak anlamlı bir farklılık saptanmadı. Ancak TSH değeri AA'lı grupta istatistiksel olarak anlamlı yüksek bulundu (p=0,03). Tartışma: Çalışmamız bölgemizdeki alopesi areata tanılı hastaların demografik, klinik ve laboratuar özelliklerini göstermektedir. Ülkemizde yapılan diğer çalışmaların sonuçlarına benzer olmakla beraber farklılıklar sapanmıştır.

#### Anahtar Kelimeler

Alopesi Areata; Demografik Özellikler; Klinik Özellikler; Laboratuvar Özellikler

#### Abstract

Aim: Alopecia areata is a chronic inflammatory disease; characterized with patchy hair loss and occurs in people of all ages and both gender. This study aims to investigate the demographic characteristics of the patients with alopecia areata in our region. Material and Method: Data of 246 patients who were diagnosed as alopecia areata in University of Gaziosmanpasa, Medical Faculty, Department of Dermatology, between January 2013 and January 2015 were evaluated retrospectively. 87 age and gender mathched subjects were enrolled as control group. Results: 246 patients, 101 female (41,1%) and 145 male (58,9%) were included in the study. Mean age of the all patients were 29,84±10,92. 31 patients were <18, 89,4% were <40 and 67,5% were 20-40 years old. 240 patients (97,5%) had AA, 5 (2%) had alopecia totalis, 1 had (0,4%) alopecia universalis. 23% of the patients were diagnosed in the age of smaller than 20. 95,9% of the patients had mild involvement in the scalp and 4,1% had severe. 69 patients had (28%) nail involvement. There were not a significant difference between disease severity in scalp and nail involvement, gender and disease initiation age (p>0,05). 18 patients (7,3%) had family history. Initiation age and severity of the disease and family history had not a significant difference (p>0,05). Free T3, free T4, hemogram, ferritin, vitamin B12 and folat levels of the patients and the controls were not significantly different. But TSH levels were significantly high in AA group (p=0,03). Discussion: Our study shows the demographic characteristics of the alopecia areta patients in our region. The results are similar to other studies in our country, however, some differences has been estimated.

#### Keywords

Alopecia Areata; Demographic Characteristics; Clinical Characteristics; Laboratory Characteristics

DOI: 10.4328/JCAM.3788 Received: 22.07.2015 Accepted: 22.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 606-8 Corresponding Author: Havva Yıldız Seçkin, Department of Dermatology, Gaziosmanpasa University School of Medicine, 60100 Tokat, Turkey. GSM: +905057505216 E-Mail: havvayildiz1982@mynet.com

#### Giris

Alopesi areata (AA), kıl foliküllerini hedef alan, skarsız kıl kaybı ile seyreden yamalarla karakterize, yaygın ve otoimmün bir hastalıktır [1]. Genel popülasyonun %0.1-0.2'sini etkilemekle beraber her iki cinste ve her yaşta görülebilmektedir [2]. Etiyolojisi kesin olarak bilinmemektedir. Ancak infeksiyonlar, oxsidadif stres, psikolojik etkenler, genetik yatkınlık otoimmün ve nörolojik faktörlerin önemli bir rol oynadığı bilinmektedir. Saç, sakal, kaş ve kirpiklerin yanı sıra tüm vücut kılları etkilenebilir [1-3]. AA tüm etnik gruplarda görülebilen yaygın bir hastalıktır. Çalışmamızın amacı bölgemizdeki AA'lı hastaların klinik, demografik ve laboratuvar özelliklerini incelemek, ayrıca hastalık şiddeti ile klinik bulgular arasındaki ilişkileri değerlendirmektir.

### Gereç ve Yöntem

Deri ve Zührevi Hastalıkları Kliniğimize Nisan 2013- Nisan 2015 tarihleri arasında başvuran AA tanısı almış 145 erkek 101 kadın toplam 246 hastanın verileri retrospektif olarak incelendi. Hastalardan ≤ 18 yaşında olanlar çocuk hasta olarak değerlendirildi. Hastaların yaş, cinsiyet, hastalık süresi, hastalığın başlama yaşı, alopesi tutulum alanları (saç, sakal, kaş, kirpik, diğer vücut kılları), aile öyküsü, atak sayısı (<4 ve ≥4) tırnak tutulumu ("pitting", longitudinal çizgilenme, lökonişi, transvers çizgilenme), eşlik eden emosyonel stres hikayesi, tiroid fonksiyon testleri (serbest T3, serbest T4, TSH), hemogram, ferritin, vitamin B12 ve folat değerleri kaydedildi. Olgular lezyon yaygınlığına göre hafif şiddette olanlar (saçlı deri tutulumu <%50, grup 1) ve şiddetli olanlar (saçlı deri tutulumu >%50, grup 2) olmak üzere iki gruba ayrıldı. Hastalık süreleri ≤ 1yıl ve > 1yıl olarak sınıflandırıldı. Hastalar klinik olarak AA, alopesi totalis ve alopesi üniversalis olarak ayrıldı. Hastaların laboratuar özelliklerini değerlendirmek ve karşılaştırmak için başka nedenlerle polikliniğimize başvuran, AA tanısı almayan ve sistemik hastalığı olmayan bireylerden oluşan, yaş ve cinsiyet açısından uyumlu 89 kontrol grubu poliklinik hasta kayıt dosyalarından retrospektif olarak taranarak çalışmaya dahil edildi.

Bulgular SPSS (18,0) istatistik programına kaydedildi. Değişkenler arasındaki ilişkileri belirlemede Ki-kare testi kullanıldı ve hesaplamalarda istatistik anlamlılık düzeyi p<0,05 olarak alındı.

#### Bulgular

Hastaların 101'i kadın (%41,1) ve 145'i erkekti (%58,9). Hastaların yaş ortalaması 29,84±10,92 idi. Hastaların 31'i çocuk (<18 yaş), 215'i erişkin (≥18 yaş), %89,4'ü 40 yaş altında ve %67,5'i 20-40 yaş arasındaydı. Hastaların 240'ında (%97,5) AA, 5'inde (%2) alopesi totalis, 1'inde (%0,4) alopesi üniversalis mevcuttu. Hastalarda hastalığın başlangıç yaşı 2-52 yaş arasında değişmekte olup, kadınlarda ortalama 28,6±11,2 yıl ve erkeklerde ise 27,8±11,2 olarak tespit edildi. Hastaların %23'ünde hastalığın başlangıç yaşının 20 yaşın altında olduğu saptandı. Hastaların cinsiyetleri ile hastalık başlangıç yaşları (çocukluk ve erişkinlik dönemi) arasında yapılan karşılaştırma sonrası istatistiksel olarak anlamlı bir ilişki bulunmadı (p>0,05). Ayrıca AA'nın şiddetiyle hastalık süresi arasında istatistiksel olarak anlamlı bir ilişki bulunmadı (p>0,05).

Tutulum alanına bakıldığında 190 hastada (%77,2) saçlı deri, 49 hastada sakal (%19,9), 4 hastada (%1,6) kaş, 2 hastada (%0,8) kirpik ve 1 hastada (%0,4) diğer vücut kıllarında dökülme (ak-

silla, ekstremiteler, pubik bölge) mevcuttu. Hastaların atak sayısı değerlendirildiğinde; %90,6'sinin atak sayısı 4'ün altında, %9,4'ünün atak sayısı 4 ve üstünde saptandı. Hastalarda tırnak tutulumu değerlendirildiğinde, 69 hastada (%28) tırnak tutulumu tespit edildi. Tırnak tutulumu olan hastalardan 13 hastada (%5,3) pitting, 21 hastada (%18,7) longitudinal cizgilenme ve 10 hastada (%4,1) lökonişi tespit edildi. Hastaların %95,9'unda hafif tutulum ve %4,1'inde siddetli tutlum vardı. Saçlı deride hastalık şiddeti ve tırnak tutulumu karşılaştırıldığında istatistiksel olarak anlamlı bir ilişki saptanmadı (p>0,05).

Hastaların %53,7'sinin öyküsünde eşlik eden emosyonel stres hikayesi varken %46,3'ünde yoktu. Hastaların %5,3'ünde fokal enfeksiyon eşlik ediyordu. Hastaların 18'inde (%7,3) aile hikayesi mevcuttu. Aile hikayesi ile hastalığın başlangıç dönemi arasında anlamlı bir ilişki saptanmadı (p>0,05). Saçlı derideki hastalık şiddeti ile aile hikayesi arasında anlamlı bir ilişki bulunmadı (p>0,05). Eşlik eden hastalık öyküsünde ise 2 hastada (%0,8) tip2 divabet 3 hastada (%1,2) hepatit B, 8 hastada tiroidit (%3,3) ve 2 hastada astım (%0,8) olduğu saptandı. Hastaların klinik ve demografik özellikleri tablo 1 de gösterildi.

Hastaların laboratuar özellikleri kontrollerle karşılaştırıldığında, serbest T3, serbest T4, hemogram, ferritin, vitamin B12 ve folat değerleri açısından istatistiksel olarak anlamlı bir farklılık saptanmadı. Ancak TSH değeri AA'lı grupta istatistiksel olarak anlamlı yüksek bulundu (p=0,03). Hastaların laboratuvar özellikleri kontrollerle karşılaştırılmalı olarak tablo 2 de gösterildi.

Tablo 1. Hastaların klinik ve demografik özellikleri

	n=246	%
Cinsiyet, kadın/erkek	101/145	41,1/58,9
Yaş	29,84±10,92	
Başlangıç yaşı (yıl)		
Kadın/ erkek	28,6±11,2 /27,8±11,2	
Atak sayısı		
İlk atak	186	75,6
2-3 atak	36	14,7
≥4	23	9,7
Saçlı deri şiddeti		
Hafif( <%50)	236	95,6
Şiddetli (≥%50)	10	4,1
Tutulum alanı		
Saçlı deri	182	73,9
Sakal	49	19,4
Kaş	4	1,6
Kirpik	1	0,4
Diğer	1	0,4
Tırnak tutulumu		
Pitting	13	5,3
Longitudinal çizgilenme	46	18,7
Lokönişi	10	4,1
Aile hikayesi		
Var	18	7,3
Yok	228	93,7

#### **Tartisma**

Tüm dünyada yaygın olarak görülen AA her iki cinste ve her yaşta görülebilmektedir. Yama tarzında saç kaybı ile karakterize

Tablo 2. Hastaların laboratuar özelliklerinin kontrollerle karşılaştırılması

	Alopesi Areata(n=246)	Kontrol (n=89)	p değeri
B12 vitamini	272,9±9,2	272,7±18,5	p>0.05
Ferritin	60,5±4,1	52,2±8,1	p>0.05
Folat	7,9±0,1	7,4±0,3	p>0.05
Hb	14,6±0,1	13,9±0,2	p>0.05
sT4	1,1±0.02	1.2±0.02	p>0.05
sT3	3,4±0,05	3,4±0,07	p>0.05
TSH	1,96±0,08	1,51±0,1	p<0.05

otoimmun bir hastalıktır ve dermatoloji polikliniğine başvuran hastaların %0,7-3,8' ini oluşturmaktadır [4]. Her iki cinste eşit olarak görülmesine rağmen bu oran bölgesel değişiklik göstermektedir. Ülkemizde yapılan çalışmaların bazılarında kadınlarda bazılarında ise erkeklerde daha sık olarak saptanmıştır. Yunanistan, Fransa, İtalya, İspanya gibi Akdeniz ülkelerinde yapılan çalışmalarda erkeklerde daha sık, İngiltere' de yapılan çalışmalarda ise kadınlarda daha sık olarak bildirilmiştir [2,5-10]. Çalışmamızda ise bölgemizde erkeklerde görülme sıklığının biraz daha fazla olduğu belirlenmiştir.

AA her yaşta görülebilse de 40 yaş altında daha yaygın olduğu ve başlangıç yaşının sıklıkla yaşamın ilk 3 on yılında olduğu gösterilmiştir [11]. Çalışmamızda literatürle uyumlu olarak hastaların %89,4'nün 40 yaş altında ve %67,5'nin 20-40 yaş arasında olduğu saptanmıştır. Yorgancılar ve arkadaşlarının 100 AA tanılı hastada yaptıkları çalışmalarında hastaların %64'ünde başlangıç yaşını 20 yaş altında tespit etmiştir [12]. Çalışmamızda ise bu oran %23 olarak bulunmuştur. Hastalığın başlangıç dönemi ile cinsiyeti karşılaştırıldığında ülkemizde ve Hindistan' da yapılan çalışmalarda çoçukluk döneminde hastalık başlangıçının kadınlarda erkeklere göre daha yüksek, Yunanistan' da yapılan bir çalışmada ise erkeklerde daha yüksek olduğu tespit edilmiştir [2,5,10]. Çalışmamızda ise hastaların cinsiyetleri ve hastalık başlangıç dönemleri arasında anlamlı bir farklılık bulunmamıştır. Ayrıca cinsiyet ile hastalık şiddeti arasındaki ilişki karşılaştırıldığında Çin ve Singapur'da yapılan çalışmalarda erkeklerde daha şidettli olduğu gösterilmiştir [7,13]. Ancak ülkemizde yapılan çalışmalarda herhangi bir ilişki gösterilememiştir [5,6,12]. Çalışmamızda da cinsiyet ile hastalık şiddeti arasındaki bir ilişki saptanmadı.

AA tüm kıllı deriyi etkilese de en sık saçlı deri sonrasında sakal bölgesi ve diğer alanlar etkilenmektedir [14]. Çalışmamızda literatürle uyumlu olarak hastaların %73,9'unda saçlı deri ve %19,9'unda sakal tutlumu saptanmıştır. AA'nın tip 2 diyabet, pemfigus foliaseus, vitiligo, tiroid hastalıkları ve romatoid artrit gibi multiple otoimmün hastalıklarla birlikteliği bilinmektedir [15,16]. Tiroid hastalıklarının AA ile birlikteliği yaklaşık %1-%20 'dir [10,16]. Hastalarımızın serbest T3 ve T4 değerleri kontrol grubuyla karşılaştırıldığında istatistiksel olarak anlamlı bir ilişki saptanmazken TSH değeri istatistiksel olarak anlamlı yüksek çıkmıştır. Hastaların %3,3'ünde tiroidit ve %0,8'inde tip 2 diyabet saptanmıştır. Bu sonuçlar daha önce yapılan çalışmalarla benzer bulunmuştur.

AA'nın serum ferritin, vitamin B12, folat ve hemoblobin değerleri arasında ilişkiyi araştıran çalışmalar mevcuttu. Bu çalışmaların bir kısmında AA' lı hastlarda serum ferritin, vitamin B12, folat ve hemoblobin değerleri düşük olarak saptanmıştır. Ancak bazılarında ise istatistiksel olarak anlamlı bir ilişki gösterilememiş-

tir [17-20]. Çalışmamızda da AA ile serum ferritin, vitamin B12, folat ve hemogblobin değerleri arasında istatistiksel olarak anlamlı bir ilişki saptanmadı.

Sonuc olarak bölgemizdeki AA'lı hastaların klinik, demografik ve labarotuvar verileri diğer çalışmalar ile uyumludur. Bu verilerin ülkemize ait genel verilerin oluşturulmasına katkı sağlayacağını ve daha fazla sayıda hasta içeren çok merkezli epidemiyolojik çalışmalarla desteklenmesi gerektiğini düşünüyoruz.

#### Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı beyan edilmemiştir.

#### Kaynaklar

- 1. Alexis AF, Dudda-Subramanya R, Sinha AA. Alopecia areata: Autoimmune basis of hair loss. Eur J Dermatol 2004;14(6):364-70.
- 2. Kyriakis KP, Paltatzidou K, Kosma E, Sofouri E, Tadros A, Rachioti E. Alopesia areata prevalance by gender and age. J Eur Acad Dermatol Venereol 2009:23(5):572-3.
- 3. Wang E, McElwee KJ. Etiopathogenesis of alopecia areata:why do our patients get it? Dermatol Ther 2011; 24(3): 337-47.
- 4. Gilhar A, Etzioni A, Paus R: Alopecia areata. N Eng J Med 2012;366(16):1515-25. 5. Kılınç I, Alper S, Ceylan C, Ünal İ. Alopesi areatalı olgularda hasta profili: Bir retrospektif çalışma. Ege Tıp Dergisi 2002;41 (1):25-7.
- 6. Kavak A, Yeşildal N, Parlak AH, Gökdemir G, Aydoğan I, Anul H, et al. Alopecia areata in Turkey: demographic and clinical features. J Eur Acad Dermatol Venereol 2008;22(8):977-81.
- 7. Tan E, Tay YK, Goh CL, Chin Giam Y. The pattern and profile of alopecia areata in Singapore--a study of 219 Asians. Int J Dermatol 2002;41(11):748-53.
- 8. Guzmán-Sánchez DA. Villanueva-Quintero GD. Alfaro Alfaro N. McMichael A. A. clinical study of alopecia areata in Mexico. Int J Dermatol 2007;46(12):1308-10.
- 9. Goh C, Finkel M, Christos PJ, Sinha AA. Profile of 513 patients with alopecia areata: associations of disease subtypes with atopy, autoimmune disease and positive family history. J Eur Acad Dermatol Venereol 2006;20(9):1055-60.
- 10. Sharma VK, Dawn G, Kumar B. Profile of alopecia areata in Northern India. Int J Dermatol 1996;13(5):22-7.
- 11. Blaumeiser B, van der Goot I, Fimmers R, Hanneken S, Ritzmann S, Seymons K, et al. Familial aggregation of alopecia areata. J Am Acad Dermatol 2006:54(4):627-32
- 12. Yorgancılar S, Anlı RA, Abdioğlu RZ, Arıca M. Alopesi areatalı hastaların klinik ve demografik özellikleri. Turkderm 2013;47(3):155-7.
- 13. Yang S, Yang J, Liu JB, Wang HY, Yang Q, Gao M, et al. The genetic epidemiology of alopecia areata in China. Br J Dermatol 2004;151(1):16-23
- Berker DAR, Messenger AG, Sinclair RD: Disorders of hair. In: Rook's Textbook of Dermatology. Burns T, Breathnach S, Cox N, Griffiths C, eds. 7th ed. Oxford: Blackwell Science, 2004;63.p.1-63.
- 15. McDonagh AJ, Messenger AG. The pathogenesis of alopecia areata. Dermatol Clin 1996:14(4):661-70.
- 16. Goh C, Finkel M, Christos PJ, Sinha AA. Profile of 513 patients with alopecia areata: associations of disease subtypes with atopy, autoimmune disease and positive family history. J Eur Acad Dermatol Venereol 2006;20(9):1055-60.
- 17. Gönül M, Gül Ü, Pişkin E, Çakmak SK, Soylu S, Kılıç A ve ark. Alopesi aretalı hastaların geriye dönük değerlendirilmesi. Turk J Dermatol 2011;5:43-7.
- 18. White MI, Currie J, Williams MP. A study of the tissue iron status of patients with alopecia areata. Br J Dermatol 1994;130(2):261-3.
- 19. Boffa MJ, Wood P, Griffithes CE. Iron status of patients with alopecia areata. Br J Dermatol 1995(4);132:662-4.
- 20. Atsü AN, Karakayalı G, Allı N, Güngör E. Alopesi areatalı hastalarda hemoglobin, hematokrit ve serum ferritin düzeyleri. Turkiye Klinikleri J Dermatol 1998;8(3):121-4.

#### How to cite this article:

Seçkin H.Y, Baş Y, Takçı Z, Sezgin S. Clinical, Demographic and Laboratory Features of Patients with Alopecia Areata in Tokat Region. J Clin Anal Med 2015;6(suppl 5):

## **Evaluation of Urinary Tract** Infections Due to Candida Species



Kandidüri / Candiduria

Yeşer Karaca Derici, Sevgi Yılmaz Hancı, Arzu Bayram, M. Cem Şirin, Pınar Şamlıoğlu, Neval Ağuş, Nisel Yılmaz Tıbbi Mikrobiyoloji Laboratuvarı, İzmir Tepecik Eğitim ve Araştırma Hastanesi, İzmir, Türkiye

Çalışmamız 12-16 Kasım 2014 tarihleri arasında Antalya'da yapılan "36. Türk Mikrobiyoloji Kongresi'nde" poster olarak sunulmuştur.

#### Özet

Amaç: Üriner sistem enfeksiyonları sıklıkla bakteriler tarafından oluşturulmakla birlikte, enfeksiyonların önemli bir kısmında fungal etiyoloji saptanmakta, bunlar arasında da kandidalar ilk sırayı almaktadır. Bu çalışmada hastanemiz idrar örneklerinden izole edilen Candida türlerinin dağılımının belirlenmesi amaçlandı. Gereç ve Yöntem: Candida tür tayini germ tüp testi, chrom agar Candida (Biomerieux, France)'daki koloni rengi ve API ID32C AUX (Biomerieux, France) ticari kiti ile yapıldı. Veriler SPSS 15.0 veri analiz programı ile analiz edildi. Bulgular: Mart 2011-Mart 2014 tarihleri arasında değerlendirilen 109662 idrar kültürünün 24364 (%22)'ünde anlamlı üreme saptandı. Anlamlı üreme saptananların 1096 (%4.5)'sında maya izole edildi. Çalışmamızda en sık C. albicans (%50.5), C. tropicalis (%15.9), C. glabrata (%12.7), C. parapsilosis (%7.2), C. kefyr (%5.8), C. krusei (%5.5) türleri izole edildi. En fazla üreme anestezi yoğun bakım ünitesinde gözlendi. Tartışma: Çalışmamızda idrar örneklerinde en sık izole edilen maya türü C. albicans olarak bulundu. Hastanelerde Candida tür tayininin yapılıp, klinik dağılımlarının belirlenmesi; tedaviye yön verilmesi ve alınacak önlemler açısından oldukça önem-

#### Anahtar Kelimeler

Üriner Sistem Enfeksiyonu; Kandidüri; C. albicans

#### Abstract

Aim: Although urinary tract infections often caused by bacteria, fungal etiology is detected in a significant number of infections in which Candida is the leading cause. In this study we aimed to evaluate the distribution of Candida strains isolated from urine samples in our hospital. Material and Method: Candida species were identified based on germ tube test, colony morphology on chrom agar Candida (Biomerieux, France) and API ID32C AUX (Biomerieux, France) commercial kit. Data were analyzed with SPSS 15.0 software for data analysis. Results: During March 2011-March 2014 a total of 109662 urine cultures were evaluated and 24364 samples revealed significant growth. Of the significant growth detected 24364 (22%) samples 1096 (4.5%) were defined as yeasts. The isolates most frequently detected in this study were C. albicans (50.5%), C. tropicalis (15.9%), C. glabrata (12.7%), C. parapsilosis (7.2%), C. kefyr (5.8%), C. krusei (5.5%). The highest yeast growth was observed in anesthesia intensive care unit. Discussion: In our study, the most frequently isolated species of yeast in the urine was C. albicans. Determination of Candida species and their clinical distributions in hospitals is very important in terms of giving direction to the treatment and measures to be taken.

Urinary Tract Infection; Candiduria; C. albicans

Received: 28.06.2015 Accepted: 26.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 609-11 DOI: 10.4328/JCAM.3706 Corresponding Author: Yeşer Karaca Derici, 156 Sok. No: 5/13, Bornova, İzmir, Türkiye. T.: +90 2324890909/1791 GSM: +905327452545 E-Mail: yeserka@hotmail.com

#### Giris

Üriner sistem enfeksiyonları (ÜSE) en sık görülen nozokomiyal enfeksiyonlardır. Sıklıkla bakteriler tarafından oluşturulmakla birlikte bu enfeksiyonların %10-15'inde fungal etiyoloji saptanmakta, bunlar arasında da Candida türleri ilk sırayı almaktadır [1-2]. Hastanede yatan hastalarda kandidüri insidansının son 10 yılda %1'den %8'e yükseldiği bildirilmiştir [3]. Özellikle geniş spektrumlu antibakteriyel ilaçların, kortikosteroidlerin ve immünsüpresif ajanların kullanımı ile birlikte uzun süreli üriner kateterizasyonun bu yükselişte önemli rolü bulunmaktadır [4]. Kandidürilerde en sık etken C. albicans olmakla birlikte, bunun dışındaki türlerin de sıklığı giderek artmaktadır [5].

Bu çalışmada idrar örneklerinden izole edilen Candida türlerinin tayini ve kliniklere göre dağılımının belirlenmesi amaçlanmıştır.

#### Gerec ve Yöntem

Hastanemiz mikrobiyoloji laboratuvarında Mart 2011-Mart 2014 tarihleri arasında değerlendirilen idrar kültürlerinde üreyen mantarların tür tayini yapıldı. Üriner sistemin fungal enfeksiyonlarında bir eşik değer belirlenmediğinden herhangi bir konsantrasyonda Candida üremesi anlamlı kabul edildi.

Hastanemiz bakteriyoloji laboratuvarına gönderilen idrar örnekleri rutin izolasyon amacıyla %5 koyun kanlı ve EMB besiyerlerine (Salubris, Türkiye) ekilip 37°C'de 24-48 saat inkübe edilerek üriner patojenler yönünden incelendi. Maya mantarı üreyen örnekler tür tayini için mikoloji laboratuvarına gönderildi. Bu örnekler mısır unu Tween 80 agar (Salubris, Türkiye), Sabouraud dekstroz agar (Salubris, Türkiye) ve chrom agar Candida (Biomerieux, France) besiyerlerine ekildi. Mayalar germ tüp oluşturması, mikroskobik morfolojileri ve koloni rengine göre değerlendirildi. Bu yöntemler ile tür tayini yapılamayanlar karbonhid-

rat asimilasyon özellikleri için API ID 32C (BioMerieux, Fransa) kitiyle değerlendirildi. Veriler SPSS 15.0 veri analiz programı ile analiz edildi.

#### Bulgular

Değerlendirilen 109662 idrar kültürünün 24364 (%22.2)'ünde üreme saptandı, bunların 1096 (%4.5)'sında maya mantarı izole edildi. Olguların 244 (%22.3)'ü toplum kökenli, 852 (%77.7)'si hastane kökenli idi. İdrar kültürlerinde maya mantarı üreyen olguların 459 (%41.9)'u erkek, 637 (%58.1)'si kadındı. Olguların yaş ortalaması 55,91±27,16 olarak bulundu. Çalışmamızda C. albicans (%50.5), C. tropicalis (%15.9), C. glabrata (%12.7), C. parapsilosis (%7.2), C. kefyr (%5.8) ve C. krusei (%5.5), C. lusitaniae (%0.9), Trichosporon spp. (%0.5), C. famata (%0.3), C. inconspicua (%0.3), C. dubliniensis (%0.1), C. guilliermondii (%0.1), C. pelliculosa (%0.1) ve C. utilis (%0.1) izole edilen türler olarak saptandı (Tablo 1).

Üremeler klinik dağılımına göre değerlendirildiğinde en fazla üremenin anestezi yoğun bakım ünitesinde olduğu gözlendi. C. albicans en sık anestezi, beyin cerrahisi, pediatri, nöroloji ve iç hastalıkları yoğun bakım üniteleri; pediatri, genel cerrahi, iç hastalıkları, enfeksiyon hastalıkları, acil servis ve üroloji kliniklerinde; C. kefyr böbrek nakli biriminde; C. tropicalis ise kalp damar cerrahisi yoğun bakım ünitesi ve nöroloji kliniğinde üredi (Tablo 2).

Kandidüri saptanan olguların 248 (% 22.6)'inde böbrek yetmezliği, 164 (% 14,9)'ünde diyabetes mellitus, 796 (%72.6)'sında idrar kateteri ve 510 (%46.5)'unda geniş spektrumlu antibiyotik kullanımı mevcuttu. Kandidüri için risk faktörleri Tablo 3'de gösterilmiştir.

Tablo 1. İdrar örneklerinden izole edilen maya türlerinin dağılımı

Maya türleri	Sayı	%
C. albicans	553	50,5
C. tropicalis	175	15,9
C. glabrata	139	12,7
C. parapsilosis	79	7,2
C. kefyr	64	5,8
C. krusei	60	5,5
C. lusitaniae	10	0,9
Trichosporon spp.	6	0,5
C. famata	3	0,3
C. inconspicua	3	0,3
C. dubliensis	1	0,1
C. guillermondi	1	0,1
C. pelliculosa	1	0,1
C. utilis	1	0,1
Toplam	1096	100,0

Tablo 2. Örneklerin kliniklere ve soyutlanan Candida türlerine göre dağılımı

Table 2. Strickler in Kill	C. albicans n (%)		C. parapsilosis n (%)	C. glabrata n (%)	C. kefyr n (%)	C. krusei n (%)	Diğerleri* n (%)
Yoğun Bakım							
Anestezi (193)	101 (52.3)	58 (30.1)	8 (4.1)	8 (4.1)	8 (4.1)	3 (1.6)	7 (3.7)
Pediatri (39)	30 (76.9)	1 (2.6)	1 (2.6)	2 (5.1)	1 (2.6)	-	4 (10.2)
İç hastalıkları (127)	73 (57.5)	15 (11.8)	13 (10.2)	15 (11.8)	5 (3.9)	3 (2.4)	3 (2.4)
Nöroloji (78)	47 (60.3)	10 (12.8)	9 (11.5)	8 (10.3)	3 (3.8)	1 (1.3)	-
Kalp Damar Cer (3)	1 (33.3)	2 (66.7)	-	-	-	-	-
Beyin Cerrahisi (9)	7 (77.8)	-	1 (11.1)	-	1 (11.1)	-	-
Koroner (19)	7 (36.8)	1 (5.3)	2 (10.5)	6 (31.6)	-	3 (15.8)	-
Böbrek nakli (7)	2 (28.6)	1 (14.3)	-	-	3 (42.8)	1 (14.3)	-
Klinikler							
Genel Cerrahi (10)	5 (50)	3 (30)	-	2 (20)	-	-	-
Pediatri (84)	48 (57.1)	9 (10.7)	9 (10.7)	6 (7.1)	4 (4.7)	6 (7.1)	2 (2,6)
İç hastalıkları (195)	87 (44.6)	28 (14.4)	8 (4.1)	36 (18.5)	19 (9.7)	14 (7.2)	3 (1.5)
Enfeksiyon Hst (107)	48 (44.9)	13 (12.1)	10 (9.3)	22 (20.6)	6 (5.6)	6 (5.6)	2 (1.9)
Üroloji (139)	47 (33.8)	25 (18)	11 (7.9)	21 (15.1)	10 (7.2)	20 (14.4)	5 (3.6)
Ortopedi (4)	2 (50 )	1 (25)	1 (25)	-	-	-	-
Nöroloji (5)	2 (40)	3 (60)	-	-	-	-	-
Yenidoğan (14)	7 (50)	-	5 (35.7)	1 (7.1)	1 (7.1)	-	-
Acil Servis							
Pediatri (24)	20 (83.3)	-	1 (4.2)	2 (8.3)	1 (4.2)	-	-
Erişkin (39)	19 (48.7)	5 (12.8)	-	10 (25.6)	2 (5.1)	3 (7.7)	-

<sup>\*</sup> Diğerleri: C. lusitaniae, Trichosporon spp., C. famata, C. inconspicua, C. dubliniensis, C. guilliermondii, C. pelliculosa, C. utilis

Tablo 3. Kandidüri için risk faktörleri

Risk faktörleri	Kandidüri (n)	%
Kadın cinsiyet	637	58.1
Böbrek yetmezliği	248	22.6
Diyabetes mellitus	164	14,9
Yoğun bakım ünitesi	475	43.3
İdrar kateteri	796	72.6
Geniş spektrumlu antibiyotik kullanımı	510	46.5

#### Tartışma

Hastanede vatan hastalarda üriner sistemin fungal enfeksiyonların insidansı son 20 yılda belirgin şekilde artmıştır [2,3]. İdrar kültürlerinin %5-10'unda Candida türlerinin ürediği bildirilmiştir [4,6-7]. Çalışmamızda da benzer şekilde 24.364 idrar kültürünün %4.5'inde maya mantarı izole edilmiştir.

Literatürde kadınlarda erkeklere göre daha yüksek oranlarda kandidüri görüldüğü bildirilmektedir [8-10]. Bizim çalışmamızda da olguların 637 (%58.1)'si kadın, 459 (%41.9)'u ise erkekti. Yoğun bakım üniteleri gibi riskli hastaların yattığı birimlerde kandidüri görülme oranları ise %19-44 arasında değişmektedir [6,7]. Kandidüri insidansındaki bu artışın en önemli nedenleri yoğun bakım ünitelerinde daha komplike hastaların yatması; fungal invazyonu kolaylaştıran invazif girişimlerin sıkça uygulanması; kortikosteroid, antineoplastik, immünsüpresif ajanlar ve geniş spektrumlu antibiyotiklerin uzun süreli kullanımı; 65 yaşın üzerinde olma, diyabetes mellitus ve kronik böbrek yetmezliği gibi eşlik eden metabolik hastalıkların bulunmasıdır [1,2,4,8]. Çalışmamızda kandidüri saptanan olguların 248 (% 22.6)'inde böbrek yetmezliği, 164 (% 14,9)'ünde diyabetes mellitus, 796 (%72.6)'sında idrar kateteri ve 510 (%46.5)'unda geniş spektrumlu antibiyotik kullanımı mevcuttu. Yedi (% 0.6) olguya ise böbrek nakli uygulanmıştı.

Yapılan epidemiyolojik çalışmalarda C. albicans, kandidürilerin %50'sinden sorumlu bulunmuştur. Diğer etkenler arasında C. glabrata ve C. tropicalis, C. parapsilosis, C krusei yer almaktadır [5,9]. Da Silva ve arkadaşları [11] idrar örneklerinden izole ettikleri Candida suşlarını sırasıyla C. albicans, C. tropicalis, C. glabrata ve C. parapsilosis olarak tanımlamışlardır. Jain ve arkadaşları [12], 55 hastanın idrar örneğinden izole ettikleri 67 Candida suşunu sıklık sırasına göre C. albicans, C. glabrata ve C. tropicalis olarak bildirmişlerdir. Ülkemizde ise Atalay ve arkadaşları [13], idrardan izole ettikleri Candida suşlarının %30'unu C. albicans ve C. glabrata, %23'ünü C. tropicalis olarak tanımlamışlardır. Yüksekkaya ve arkadaşları [14] yoğun bakım ünitesinde yatan hastaların idrarlarından en sık C. albicans, ikinci ve üçüncü sıklıkta ise sırasıyla C. glabrata ve C. tropicalis ürediğini saptamışlardır. Kaya ve arkadaşları [15], kandidüri etkeni olarak en sık C. albicans, sonra C. glabrata ve C. kefyr'i saptamışlardır. Yazarlar tedaviyi yönlendirmesi açısından üretilen maya kolonilerinin tür tayininin yapılmasını önermişlerdir. Çalışmamızda literatürle uyumlu olarak C. albicans, C. tropicalis ve C. glabrata sırasıyla %50.5, %16.0, %12.7 oranında saptandı. En sık üreyen maya türü yoğun bakım üniteleri, genel cerrahi, pediatri, iç hastalıkları, enfeksiyon hastalıkları ve üroloji kliniklerinde C. albicans; böbrek nakli biriminde C. kefyr, kalp damar cerrahisi yoğun bakım ünitesi ve nöroloji kliniğinde C. tropicalis olarak belirlendi. Bunun sebebinin böbrek nakli ve kalp damar cerrahisi yoğun bakım ünitesinde yatan hasta sayılarının ve gönderilen örneklerin göreceli olarak az olmasından kaynaklanabileceği düsünülmektedir.

Sonuç olarak, çalışmamızda idrar örneklerinde en sık izole edilen mava türü C. albicans'tır. Kandidaların neden olduğu ÜSE'lerin uygun tedavisi için etkenlerin tür tanımlanmasının yapılması gerekmektedir. Özellikle yoğun bakım ünitelerinde yatan hastalarda kandidüri sıklığının yüksek olduğu akılda tutulmalı ve bu hastalar yakından izlenmelidir.

#### Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı beyan edilmemiştir.

#### Kaynaklar

- 1. Phillips IR. Karlowicz MG. Prevelance of Candida species in hospital acquired urinary tract infections in a neonatal intensive care unit. Pediatr Infect Dis J 1997;16:190-4.
- 2. Febre N, Silva V, Medeiros EAS, et al. Microbiological characteristics of yeasts isolated from the urinary tract of intensive care unit patients undergoing urinary catheterization. I Clin Microbiol 1999:37:1584-6.
- 3. Durupınar B, Saniç A, Pekbay A, Günaydın M, Özdemir Ş. Hastane kaynaklı kandidüri. Mikrobiyol Bült 1996; 30(2):171-6.
- 4. Achkar JM, Fries BC. Candida infections of the genitourinary tract. Clin Microbiol Rev 2010;23(2):253-73
- 5. Sobel JD, Kauffman CA, McKinsey D, et al. Candiduria: a randomized double blind study of treatment with fluconazole and placebo. Clin Infect Dis 2000;30(1):19-24. 6. Alvarez-Lerma F, Nolla-Salas J, Leon C, et al. Candiduria in critically ill patients admitted to intensive care medical units. Intensive Care Med 2003;29(7):1069-76. 7. Passos XS, Sales WS, Maciel PJ, et al. Candida colonization in intensive care unit
- patients' urine. Mem Inst Oswaldo Cruz 2005:100(8):925-8.
- 8. Paul N, Mathai E, Abraham OC, Michael JS, Mathai D. Factors associated with candiduria and related mortality. J Infection 2007;55(5):450-5.
- 9. Mirdha BR, Sethi S, Banerjee U. Prevalence of fungal species in patients with funguria. Indian J Med Res 1998;107:90-3.
- 10. Alpat SN, Özgüneş I, Ertem OT, Erben N, Doyuk Kartal E, Tözün M, Usluer G. Kandidürisi olan hastalarda risk faktörlerinin değerlendirilmesi. Mikrobiyol Bul 2011;45(2):318-24.
- 11. Da Silva EH. Ruiz Lda S. Matsumoto FE. Auler ME. Giudice MC. Moreira D. et. al. Candiduria in a public hospital of São Paulo (1999-2004): characteristics of the yeast isolates. Rev Inst Med Trop Sao Paulo 2007;49(6):349-53
- 12. Jain N, Kohli R, Cook E, Gialanella P, Chang T, Fries BC. Biofilm formation by and antifungal susceptibility of Candida isolates from urine. Appl Environ Microbiol 2007;73(6):1697-703.
- 13. Atalay MA, Koç AN, Sav H, Demir G. Yatan hastaların idrar kültürlerinden izole edilen Candida türleri ve antifungal duyarlılıkları. Turk Hij Den Biyol Derg 2013;70(4):185-90.
- 14. Yüksekkaya Ş, Fındık D, Arslan U. Yoğun bakım ünitesinde yatan hastaların idrarlarından izole edilen Candida türlerinin moleküler epidemiyolojisi ve antifungal duyarlılıkları. Mikrobiyol Bul 2011; 45(1): 137-49.
- 15. Kaya K, Kaya S, Avunduk H, Özyazıcı G, Bakıcı MZ. Cumhuriyet üniversitesi tıp fakültesi hastanesi'nde 10 aylık periyotta saptanan kandidüri etkenlerinin dağılımı ve antifungal duyarlılıkları. C. Ü. Tıp Fak Derg 2004;26 (2): 71-4.

#### How to cite this article:

Derici Y.K, Hancı S.Y, Bayram A, Şirin M.C, Şamlıoğlu P, Ağuş N, Yılmaz N. Evaluation of Urinary Tract Infections Due to Candida Species. J Clin Anal Med 2015;6(suppl 5): 609-11.



## Urine Iodine Excretion and Iodine Deficiency Status in School Age Children at Isparta Province

## Isparta İli Okul Çağı Çocuklarında İdrar İyot Atılımı ve İyot Eksikliği Durumu

Isparta İli Okul Çağı Çocuklarında İyot Eksikliği / Iodine Deficiency Status in School Age Children at Isparta

Nagehan Aslan¹, Hüseyin Kurku², Özgür Pirgon³
¹Pediatri Ana Bilim Dalı, Süleyman Demirel Üniversitesi Tıp Fakültesi, Isparta,
²Biyokimya Ana Bilim Dalı, Konya Eğitim ve Araştırma Hastanesi, Konya,
³Pediatrik Endokrinoloji Ana Bilim Dalı, Süleyman Demirel Üniversitesi Tıp Fakültesi, Isparta, Türkiye

#### Özet

Amaç: Önlenebilir zeka geriliğinin en sık nedeni olan iyot eksikliği dünyada ve ülkemizde hala önemli bir problem olarak yerini korumaktadır. İlkokul çağı çocuklarında hafif ve orta düzeyde iyot eksikliği saptanır ve tedavi edilirse bilişsel ve motor işlevlerin düzeldiği bilinmektedir. Biz bu çalışmada endemik guatr bölgesi olan ilimizde idrar iyot seviyesi ölçümü ile iyot eksiklik düzeyini araştırdık. Çalışmamızda 7-12 yaş arası okul çağı çocuklarının idrar iyot atılımı belirlenerek iyot düzeyleri inceledik, eksiklik saptanan çocuklarda ileri tetkik ve tedavi ile zeka geriliğinin önlenmesini amaçladık. Gereç ve Yöntem: Isparta ilinde Milli Eğitim Bakanlığına bağlı beş ilkokul ve beş ortaokul olmak üzere toplam on okuldan, 7-12 yaş arası, toplam 400 çocuk çalışmaya alındı. Çocukların spot idrar örnekleri alınarak Sandell-Kolthoff reaksiyonu ile spektrofotometrik olarak iyot düzeyleri ölçüldü. Ayrıca iyotlu tuz ve hazır su kullanım durumu sorgulandı. Verilerin istatistiksel analizi SPSS 20.0 versiyonu kullanılarak yapıldı. Bulgular: Çocukların %56,7'si erkek, %43.3'ü kızdı. Ortalama yaş 8,7±1,2 yıl, ortalama boy 131,8±10,7 cm, ortalama ağırlık 29,7±7,2 kg idi. Çocukların %63,7'sinde (254) iyot eksikliği saptandı ve bunların büyük çoğunluğunda (%81,7) hafif derecede iyot eksikliği mevcuttu. İyot eksikliği saptananların %57,4'ü kız, %42,6'sı erkekti ve %85'i iyotlu tuz, %15'i iyotsuz tuz kullanmakta idi. Isparta ilinde ortalama iyot düzeyi 107,8±78,1µg/L idi. İdrar iyot düzeyi düşük olan çocuklarda antropometrik ölçümler anlamlı olarak düşüktü. Tartışma: Çalışmamızda saptadığımız bulgulara ve önceki çalışmalarla karşılaştırarak elde ettiğimiz sonuçlara dayanarak Isparta ilinde iyotlu tuz tüketimimin son yıllarda arttığını ve iyot beslenmesinin yeterli düzeyde olduğunu tespit ettik.

#### Anahtar Kelimeler

Çocukluk Çağı; İyot; İdrar; Büyüme Geriliği; Isparta

#### Abstract

Aim: Iodine deficiency is the most common cause of preventable mental retardation in the world and in our country still remains in place as a major problem. If the primary school-age children in the mild to moderate iodine deficiency is detected and treated are known to improve cognitive and motor function. The aim of this study was to determine the status of iodine deficiency in school children at Isparta province. Material and Method: The study enrolled a total of 400 students aged 7-12 without chronic disease. Children's anthropometric measurements, salt and drinking water preferences were recorded. Urinary iodine levels, was measured by spectrophotometry with Sandell-Kolthoff reaction. Results: The 56.7% of the children were male and 43.3% female. The mean age was 8.7 ± 1.2 years, mean height 131.8 ± 10.7 cm and the mean weight was 29.7  $\pm$  7.2 kg. Children in 63.7% (254) had iodine deficiency and the majority of them (81.7%) had mild iodine deficiency. 57.4% of children with iodine deficiency were female and 42.6% male. 85% of these children was using iodized salt, 15% of non-iodised salt. The mean urinary iodine level of 400 children was 107,8±78,1µg/L. Anthropometric measurements in children with urinary iodine levels were significantly lower. Discussion: In our study we determined mild iodine deficiency in primary scholl age children in Isparta and we have found a statistically significant growth retardation in children with iodine deficiency.

#### Keywords

Childhood; Iodine; Urine; Growth Retardation; Isparta

DOI: 10.4328/JCAM.3845 Received: 22.08.2015 Accepted: 08.09.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 612-6 Corresponding Author: Nagehan Aslan, Pediatri Anabilim Dalı, Süleyman Demirel Üniversitesi Tıp Fakültesi Hastanesi, Çünür, 32000, İsparta, Türkiye. GSM: +905055499986 E-Mail: nagehan\_aslan@hotmail.com

#### Giris

Bugün dünyada yaklaşık 2.2 milyar kişi iyot eksikliği olan bölgelerde yaşamakta ve iyot eksikliğinin komplikasyonları açısından risk altında bulunmaktadır [1]. İyot eksikliği önlenebilir zeka geriliğinin en sık nedenidir [2]. Beyin hasarı açısından en çok etkilenilen dönemler gebelik, fetal ve erken postnatal dönem olmakla birlikte iyot eksikliği her yaştaki bireyi etkilemekte ve guatr ile karşımıza çıkmaktadır. İyot eksikliği embriyonal dönemde başlarsa; sağırlık, dilsizlik, spastik dipleji, şaşılık, ağır nörolojik defisit (nörolojik kretenizm), cücelik, psikomotor gerilik, ölü doğum ve zeka geriliği gibi sağlık sorunlarına neden olmaktadır [3]. İyot başlıca toprakta olmak üzere su ve havada bulunan bir eser elementtir. Günlük iyot gereksiniminin %90'ı gıdalardan, %10'u içme suyundan sağlanır. Sağlıklı insanda gıdalardaki iyodun yaklaşık %90'ı emilmektedir [2]. Emilim mide ve bağırsaklarda olur ve yaklaşık bir saatte tamamlanır. Günlük iyot gereksinimi yaşa, fizyolojik gereksinime ve bazı hastalıklara göre farklılık gösterir. Erişkinlerde en az 1µg/kg/gün iyot alınması gerekirken, gebelik ve yenidoğan döneminde gereksinim daha fazladır. Dünya Sağlık Örgütü (DSÖ) verilerine göre değişik yaş grupları için diyetle alınması önerilen iyot miktarları Tablo 1'de gösterilmiştir.

Tablo 1. Yaşa göre alınması önerilen iyot miktarları

Yaş grubu	Diyetle alınması gereken iyot miktarı (µg/gün)
0-5 yaş	90
6-12 yaş	120
>12 yaş	150
Gebelik	250
Emzirme dönemi	250

İyot eksikliği halen gelişmekte olan ülkeler için çok önemli bir halk sağlığı sorunu olmaya devam etmekte ve çeşitli yaş gruplarına göre farklı nedenlerle ortaya çıkabilmektedir. İyot eksikliğine neden olabilen bu faktörler Tablo 2'te gösterilmiştir.

Tablo 2. İyot eksikliğine neden olabilen faktörler

Diyette iyot eksikliği

Bağırsaklardan yetersiz iyot emilimi

Malnütrisyon

Malabsorbsivon

Fekal ve üriner inorganik ivot kavbının artması

Laktasvon

Gehelik

Vücudun tiroid hormonlarına veya iyoda gereksinimin artması

Antitiroid ilaçlarla uzun süreli tedavi

İyot kaybının artması

İyot tiroid hormon sentezi için çok önemlidir. Her yaştaki bireyi etkileyebilen iyot eksikliği klinikte guatr tablosu ile karşımıza çıkmakta ve toplum sağlığı açısından ülkemizde halen önemli bir sorun teşkil etmektedir. Eğer toplumda diyetle iyot alımı yetersiz ve buna bağlı olarak idrarda iyot atılımı düşük saptanıyorsa iyot profilaksisinin uygulanması gerekmektedir [4].

Günlük alınan iyodun yaklaşık %85-90'ı idrar ile atılmaktadır. Bu nedenle idrar iyot düzeyi o bölgedeki iyot durumunu yansıtan önemli bir kriterdir [5]. Okul çocuklarında spot idrarda iyot ölçümünde; 10µg/dl'nin altındaki değerler iyot alımında yetersizliğin göstergesidir. Yenidoğan bebeklerde 5. günden itibaren idrar iyodu ölçülebilir ve 5µg/dl'nin üzerindeki değerler normaldir.

İyot eksikliğinin prevalansı ve ağırlığını saptamada en uygun ve güvenilir yöntem idrar iyot düzeyi ölçümüdür. İdrar iyot düzeyi ve iyot alımı arasındaki ilişki tablo-3'te gösterilmektedir.

Tablo 3. İyot beslenmesi göstergesi olarak idrar iyot konsantrasyonu

Median idrar iyodu (μg/dL)	İyot durumu
<20	Ağır iyot eksikliği
20-49	Orta iyot eksikliği
50-99	Hafif iyot eksikliği
100-199	Optimal
200-299	İyotla indüklenmiş hipertiroidizm riski
300	Yan etki riski

Vücutta endokrin bezler içinde hormon sentezi için iyoda gereksinimi olan tek endokrin bez tiroiddir ve tiroid bezi iyot metabolizmasında önemli rol oynar. İyot eksikliğinin tiroid işlev ve hacmini etkilediği bilinmektedir. Endemik guatr, epidemiyolojik açıdan, tiroid hiperplazisinin belli bir coğrafi bölgede yoğunlaşmasıdır. Herhangi bir yerleşme bölgesinde, çocuk yaş grubunun (5-12 yaş) yüzde beşinde tiroid büyüklüğü (guatr) varsa endemi söz konusudur [6]. Guatr, iyot eksikliği dışında birçok çevresel ve nutrisyonel faktörlerin etkisiyle ortaya çıkabilmekle birlikte iyot eksikliği en önemli sebeptir ve diğer faktörlerin guatrojenik etkileri iyot eksikliği zemininde belirginleşmektedir [7]. Isparta ve çevresinin endemik guatr bölgesi olduğu bilinmekte ve bu durumun bölgenin coğrafi yapısının, basit guatr oluşmasında en önemli etyolojik faktör olan iyot eksikliğine yol açmasından kaynaklandığı tahmin edilmektedir.

İlkokul çağı çocuklarında hafif ve orta düzeydeki iyot eksikliği tedavi edilirse bilişsel ve motor işlevlerin düzeldiği belirtilmektedir. Bu amaçla bölgemizde öğrenim gören ilköğretim çağındaki çocukların iyot eksikliği prevalansını incelemek amacıyla beş ilkokul ve beş ortaokulda, 7-12 yaşlarındaki, 400 öğrencide bir tarama çalışması gerçekleştirdik.

#### Gereç ve Yöntem

Çalışmaya İsparta ilinde, sosyoekonomik açıdan farklı mahallelerden seçilen, 5 ilkokul ve 5 ortaokuldan, her ilkokuldan rastgele seçilen 7-10 yaş arası 40 öğrenci (toplam:200), her ortaokuldan 11-12 yaş arası rastgele seçilen 20 (toplam:200) çocuk olmak üzere toplam 400 öğrenci alındı. Tarama için İl Milli Eğitim Müdürlüğünden ve İl Sağlık Müdürlüğünden gerekli izinler alındı. Okullardaki tüm velilere taramamızın amacını ile içme suyu tercihi ve ailelerin tuz tercihi (iyotlu, iyotsuz) sorularını içeren bir anket gönderildi ve yazılı izinlerini alındı. Çocukların boy ve kilo ölçümleri kaydedildi. Taramaya katılmayı kabul etmeyen velilerin çocukları çalışmadan çıkarıldı.

Öğrencilerden kapalı plastik kaplara, hijyenik koşullarda, sabah ilk idrar örnekleri alındı. Bu örneklerin 5ml'lik kısmı deiyodinize test tüplerine ayrılıp parafinle kapatıldıktan sonra ışık geçirmez kaplara konularak aynı gün derin dondurucuda dondurularak iyot olçümüne kadar -18 Co'de saklandı. İdrar örnekleri daha sonra Ankara BioLab Laboratuarlarında Sandell-Kolthoff reaksiyonu ile spektrofotometrik olarak iyot düzeyi yönünden incelendi. Öğrencilerin boy ve kilo ölçümleri kaydedildi.

Çalışmada elde edilen bulgular değerlendirilirken, istatistiksel analizler için SPSS 20.0 programı (SPSS/Window version 20.0, Chicago, IL, USA) kullanıldı. Verilerin istatistiksel değerlendirilmesinde; ortalama, medyan ve standart derivasyon ile x2 testi, ortalamalar arasındaki farkın belirlenmesinde parametrik test olan student t testi uygulandı. P < 0.05 anlamlı olarak kabul edildi.

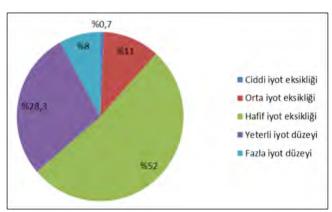
#### Bulgular

Çalışmaya 227 erkek (% 56,7) ve 173 kız (% 43,3) olmak üzere toplam 400 öğrenci alındı. Çocukların yaşları 7 ila 12 yıl arasında idi ve ortalama yaş 8,7±1,2 yıl idi. Çocukların yaş ve cinsiyet dağılımı Tablo 8'de gösterildi. Ortalama boy 131,8±10,7 cm, ortalama ağırlık 29,7±7,2 kg, ortalama vücut kitle indeksi (VKİ) 16,9±2,6 kg/m² idi.

Alınan idrar örneklerinde iyot düzeyi minimum 18,9 µg/L, maksimum 657 µg/L, ortalama idrar iyot düzeyi 107,8 $\pm$ 78,1 µg/L idi. İdrar iyot düzeyleri ciddi eksik (<20 µg/L), orta derecede eksik (20-49 µg/L), hafif derecede eksik (50-99 µg/L), yeterli iyot düzeyi (>100 µg/L), fazla iyot düzeyi (>200 µg/L) olarak derecelendirildi.

Çocukların 254'ünde (% 63,7) iyot eksikliği saptandı (Tablo 9). İyot eksikliği saptananların %57,4'ü kız, %42,6'sı erkekti (Şekil 10). İyot beslenmesi durumunun bir göstergesi olarak idrar iyot atılımını inceleyen çalışmamızda %0,7 ciddi eksiklik, %11 orta derecede iyot eksikliği, %52 hafif iyot eksikliği, %28,3 yeterli iyot düzeyi, %8 fazla iyot düzeyi saptandı (Şekil 1).

Şekil 1. İdrar iyot düzeyi dağılımı

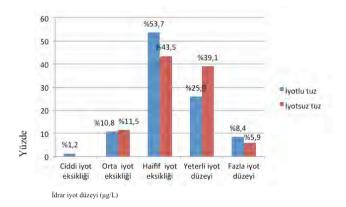


Anket sonuçlarına göre çocukların 324 tanesi (%81) iyotlu tuz, 76'sı (%19) iyotsuz tuz tüketmekteydi. İyot eksikliği saptanan hastaların 215'i (%85) iyotlu tuz, 39'u (%15) iyotsuz tuz kullanmaktaydı.

İyotlu tuz kullananların; %1,2'sinde ciddi iyot eksikliği, %10,8'inde orta iyot eksikliği, %53,7'sinde hafif iyot eksikliği, %25,9'unda yeterli iyot düzeyi, %8,4'ünde fazla iyot düzeyi saptandı. İyotsuz tuz kullananların; %11,5'inde orta iyot eksikliği, %43,5'inde hafif iyot eksikliği, %39,1'inde yeterli iyot düzeyi, %5,9'unda fazla iyot düzeyi saptandı (Şekil 2).

İyotlu tuz kullanan çocukların sayısı 324, ortalama yaşı 8,7±1,2 yıl, ortalama ağırlığı 29,7±7,2 kg, ortalama boyu 131,9±10,9 cm, ortalama VKİ 17,2±2,9 kg/m² ve ortalama idrar iyot düzeyi 122,8±83,3 µg/L idi. İyotsuz tuz kullanan gruptaki çocukların sayısı 76, ortalama yaşı 8,6±1,1 yıl, ortalama kilosu 29,8±7,3 kg, ortalama boyu 131,1±9,9 cm, ortalama VKİ 17,3±2,4 kg/m² ve ortalama idrar iyot düzeyi 106,1±83,3 µg/L idi. İyotlu tuz kullanan ve iyotsuz tuz kullanan çocukların yaş, kilo, boy, VKİ ve idrar iyot düzeyleri arasında istatistiksel olarak anlamlı farklılık sap-

Şekil 2. Tuz seçimine göre göre idrar iyot düzeyi



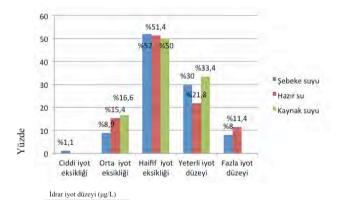
tanmadı. Bu iki grubun demografik özelliklerinin ve idrar iyot düzeyinin karşılaştırması Tablo 4'de gösterilmektedir.

İçme suyu olarak, çalışmadaki çocukların 147'si (%37) hazır su, 247'si (%62) şebeke suyu ve 6'sı (%1) kaynak suyu kullanıyordu.

Tablo 4. İyotlu tuz kullanan ve iyotsuz tuz kullanan grubun demografik özelliklerinin karşılaştırması

	İyotlu tuz kullanan grup	İyotsuz tuz kullanan grup	р
Sayı	324	76	
Yaş (Yıl)	8,7±1,2	8,6±1,1	0,880
Vücut Ağırlığı (kg)	29,7±7,2	29,8±7,3	0,983
Boy (cm)	131,9±10,9	131,1±9,9	0,602
VKİ (kg/m²)	17,2±2,9	17,3±2,4	0,645
İdrar iyot düzeyi(μg/L)	122,9±83,8	106,13±48,7	0,168

Şekil 3. İçme suyu tercihine göre idrar iyot düzeyi



Şebeke suyu kullananların; %1,1'inde ciddi iyot eksikliği, %8,9'unda orta iyot eksikliği, %52'sinde hafif iyot eksikliği, %30'unda yeterli iyot düzeyi, %8'inde fazla iyot düzeyi saptandı. Hazır su kullananların; %15,4'ünde orta iyot eksikliği, %51,4'ünde hafif iyot eksikliği, %21,8'inde yeterli iyot düzeyi, %11,4'ünde aşırı iyot düzeyi saptandı. Kaynak suyu kullananların; %16,6'sında orta iyot eksikliği, %50'sinde hafif iyot eksikliği, %33,3'ünde yeterli iyot düzeyi saptandı (Şekil 3).

İdrar iyotu eksik (<100 µg/L) olan gruptaki çocukların toplam sayısı 254, ortalama yaş 8,7±1,1 yıl, ortalama VKİ 16,7±2,6 kg/m² ve ortalama idrar iyot düzeyi 68,4±17,5 µg/L idi. İdrar iyotu yeterli (>100 µg/L) olan gruptaki çocukların toplam sayısı 146, ortalama yaş 8,8±1,3, ortalama VKİ 17,1±2,6 kg/m² ve ortala-

ma idrar iyot düzeyi 168,4±82,2 µg/L idi.

İdrar iyot düzeyi eksik olan grubun ortalama boyu 130,3±10,9 cm, idrar iyot düzeyi yeterli olan grubun ortalama boyu 133,2±10,4 cm idi ve istatistiksel olarak anlamlı idi (p=0,029). İdrar iyot düzeyi düşük olan grubun ortalama vücut ağırlığı 28,6±7,2 kg, idrar iyot düzeyi yeterli olan grubun ortalama vücut ağırlığı 30,8±7,2 kg idi ve istatistiksel olarak anlamlı idi (p=0.017). Bu iki grubun demografik ve idrar iyot düzeylerinin karşılaştırması Tablo 5'te gösterilmektedir.

Tablo 5 İvot düzevi düşük ve veterli olan grubun demografik özelliklerinin ve idrar iyot düzeylerinin karşılaştırılması

	İyot düzeyi düşük (< 100 µg/L)	İyot düzeyi yeterli (> 100 µg/L)	р
Sayı	254	146	
Yaş (Yıl)	8,7±1,1	8,8±1,3	0,051
Ağırlık (kg)	28,6±7,2	30,8±7,2	0,017
Boy (cm)	130,8±12,4	133,2±10,4	0,029
VKİ (kg/m²)	16,7±2,6	17,1±2,6	0,561
İdrar iyot düzeyi (μg/L)	68,4±17,5	168,4±82,2	0,000

#### **Tartısma**

İyot normal büyüme ve gelişmede önemli role sahip olan esansiyel bir mikrobesindir. İyot eksikliğinde temel yaklaşım kişilerin günlük iyot alımını artırmak amacı ile sık yenen besinlerin iyotla zenginleştirilmesine dayanmaktadır. Bu amaçla ülkemizde ve dünyada en sık kullanılan yöntem tuzun iyotlanmasıdır [8]. Ülkemizde 1994 yılında "İyot yetersizliği hastalıkları ve tuzun iyotlanması programı" başlatılmış ve 9 Temmuz 1998 tarih ve 23397 sayılı Resmi Gazete ile sofra tuzlarının iyotlu olarak üretilmesi zorunlu hale getirilmiştir.

Günümüze kadar Türkiye'nin iyot durumunu tam olarak yansıtan bir harita çıkarılmamış olsa da, bu konuda ülkenin çeşitli kesimlerinde çok sayıda çalışmalar yapılmıştır. Bununla birlikte İyot eksikliği prevalansını saptamada en uygun ve en güvenilir yöntem idrar iyot düzeyi ölçümüdür. Biz de bundan yola çıkarak endemik guatr bölgesi olan ilimizde iyot eksikliği ve iyotlu tuz tüketimini değerlendirmek için bir okul taraması yaptık.

Ermenistan, Tebaida ve Caimo'da merkez ve kırsalda okul çocuklarından 444 idrar numunesi alınarak yapılan çalışmada kızlar ve erkekler arasında anlamlı fark bulunamamıştır. Bu çalışmada sırasıyla %11 ve %18 oranında iyot eksikliği saptanmıştır. Bu çalışmada ekonomik düzeyi iyi olmayanlarda iyot eksikliğinin anlamlı olarak daha yüksek oranda olduğu da gösterilmiştir [9]. Bizim çalışmamızda kızlarda iyot eksikliğinin daha yüksek oranda saptanması ise kızların ergenliğe daha erken girmesine bağlı olarak iyot gereksiniminin daha erken yaşlarda artması ile ilgili olarak yorumlanmıştır.

Zimmermann MB ve ark. çalışmalarında iyotun dolaşımda IGF-1 ve IGFBP-1'i artırmak yoluyla büyüme ve gelişme üzerinde etkili olduğunu ve iyot eksikliği durumunun büyüme üzerine olumsuz etki edeceğini göstermişlerdir [10]. Bizim çalışmamızda da iyot eksikliği olan grupta boy ve vücut ağırlığı anlamlı olarak düşük tespit edilmistir.

Günümüze kadar Türkiye'nin iyot durumunu tam olarak yansıtan bir harita çıkarılmamış olsa da, bu konuda ülkenin çeşitli kesimlerinde çok sayıda çalışmalar yapılmıştır. Bununla birlikte iyot eksikliği sıklığını saptamada en uygun ve güvenilir yöntem idrar iyot düzeyi ölçümüdür. Bizim çalışmamızda da endemik guatr bölgesi olan ilimizde iyot eksikliği ve iyotlu tuz tüketimini değerlendirmek için bir okul taraması yapılmıştır.

Eser S. ve ark. 1950'li villarda 30.000 kisilik toplu guatr taramasında; Karadeniz bölgesi, İç Anadolu ve Batı Anadolu'nun iç bölgelerinde önemli ölçüde guatr bulunduğu ve ülkemizin endemik guatr kuşağında olduğu belirtilmiştir [11].

Sonraki yıllarda Koloğlu S. tarafından ülke genelinde yapılan çalışmalarda endemik bölgelerde toprak, su ve besin maddelerinde iyodun yetersiz olduğu, bu bölgeden gelen hastalarda idrar iyot düzeyinin düşük ve tiroid bezinde iyodu tutmasının aşırı olduğu bildirilmiştir. 1980-87 yılları arasında Hatemi H ve ark. tarafından tüm Türkiye'yi kapsayan 73.757 kişinin boyun palpasyonu ile taranması sonucu guatr sıklığı %30,5 olarak saptanmıştır. Bölgeler arasında Karadeniz bölgesinde guatrın en sık, Marmara bölgesinde en az olduğu görülmüştür [12] . Bizim çalışmamızda idrar iyot düzeyi ölçümü ile iyot eksikliği taranmış, tiroid fonksiyonları ve guatr değerlendirilmemiştir. Bir sonraki çalışmada iyot eksikliği saptanan çocuklarda guatr sıklığının değerlendirilmesi planladık.

İstanbul'da 1997 yılında, 3-12 yaş arası Asya ve Avrupa yakası yuva ve okul çocuklarıyla yapılan bir çalışmada %14,3 oranında guatra rastlanmış, spot idrar iyot konsantrasyonunun ortalama 227,8 ± 102,7 μg/L bulunmuş, %5,6 vakada hafif iyot eksikliği (50-100  $\mu$ g/L), %4,5'inde ise orta – ağır (<50  $\mu$ g/L) iyot eksikliği saptandığı belirtilmiştir. Bu çalışmada Asya yakasında oturanlarda idrar iyot düzeyi Avrupa yakasındakilerden anlamlı derecede düşük bulunmuştur. İstanbul'da 1998 yılında, 13-18 yaş arası 452 çocuğu kapsayan başka bir çalışmada guatr sıklığı %40, 278 yüksekokul öğrencisinde %39 olarak saptanmış ve idrar iyotu vakaların yaklaşık %20'sinde eksik bulunmuştur. Gür E. ve ark. 1999 yılında İstanbul'da yaptığı taramada öğrencilerin %46,2'sinde idrar iyotu <100 µg//L iken, 2003 yılında yine İstanbul'da Barutçugil ve arkadaşlarının taramasında bu değerin %17,8'e gerilemesi 1999 yılında zorunlu iyotlu tuz tüketimi ile ilişkilendirilmiştir [13]. Ancak Kurtoğlu S. ve arkadaşlarının çalışmasında Kayseri'de iyotlu tuz profilaksisine rağmen gebelerde ve bebeklerinde iyot eksikliği saptanmıştır [14].

Bastemir M. ve ark. Doğu Karadeniz bölgesi ve İç Anadolu bölgesinden toplam 1733 adolesanı dahil ettikleri çalışmalarında, ortalama idrar iyot düzeyi sırasıyla 139 μg//L ve 61 μg//L ölçülmüş ve iki bölgenin ortalama idrar iyot düzeyi arasında anlamlı fark saptanmıştır [15]. Kutlu R. ve ark. Konya'da 178 okuldan, 10-18 yaş arası 1847 öğrenciyi içeren çalışmasında ortalama idrar iyot düzeyi 198±46,61 µg//L saptanmıştır [16]. Darcan S. ve ark. Ege bölgesinden, 6-12 yaş arası çocuklarda yaptığı çalışmada ortalama idrar iyot düzeyi 53 µg//L iken, Budak N. ve ark. çalışmasında ise ortalama idrar iyot düzeyi 25,5±17,2 μg//L olarak bildirilmiştir [17]. Çalışmamızda ise ortalama idrar iyot düzeyi 107,8 µg /L saptanmıştır.

Erdoğan G. ve ark. 1997 yılında, iyot profilaksisi öncesinde, Isparta'da 5-12 yaş çocuklarda yaptığı çalışmada ortalama idrar iyot düzeyi 28 µg/L saptanmıştır. Benzer şekilde İsparta'da Çetin H. ve ark. 2006 yılında, 6-11 yaş arasındaki 500 çocuğu içeren çalışmasının sonuçları incelenecek olursa ortalama idrar iyot düzeyi 70 µg/L saptanmış ve çalışmaya alınan çocukların %72'sinde iyot eksikliği tespit edilmiştir [18]. Bu çalışmada Isparta ılımlı iyot eksikliği bölgesi olarak değerlendirilmiştir. Bölgede önceki yıllarda yapılan bu çalışmalarla kıyaslandığında bizim çalışmamızda ortalama idrar iyot düzeyinin 107,8 µg/L saptanması ve öğrencilerin % 63,7'sinde idrarda iyot eksikliği saptanması, bölgemizde okul çağı çocuklarında iyot beslenmelerinin son yıllarda arttığını ve idrar iyot düzeyinin yükseldiğini göstermektedir ancak 10 yıllık süreçte iyot eksikliği durumunda %72'den %63,7'ye gerileme olması elde edilen iyileşmenin yeterli olmadığını düşündürmüştür.

Eğri M. ve ark. endemik guatr bölgesi olan Malatya'da 7-11 yaş arası, 568 okul çocuğunda yaptıkları çalışmalarında ortalama idrar iyot düzeyini 66 µg/L olarak tespit etmişler [19]. Özkan B. ve ark. çalışmasında %47,6 çocukta guatr saptanmış ve ortalama tiroid hacimleri arasında cinsiyete göre anlamlı farklılık saptanmamıştır [20]. Çalışmada ortalama idrar iyot düzeyi guatrı olan grupta 20 μg/L, guatr olmayan grupta ise 50 μg/L olarak saptanmış ve bu durum istatistiksel olarak anlamlı bulunmuştur. Akpınar D. ve ark. 13-18 yaş arası, 452 öğrenciyi içeren, çalışmalarında kızlarda guatr prevalansı anlamlı yüksek saptanmış, öğrencilerin %2,9'unda nodüler guatr tespit edilmiş ve öğrencilerin %20'sinde iyot düzeyi <100 µg/L olarak ölçülmüştür. Çalışmada İstanbul'un iyot eksikliği olmayan ancak guatr için endemik bir bölge olduğu sonucu çıkarılmıştır [21].

Türkiye'nin 7 farklı bölgesinde, 24 şehirden, 30 farklı okuldan, toplam 900 öğrenci ile yapılan, 510 öğrencinin şehirde, 390 öğrencinin kırsal kesimde yaşadığı, bir çalışmada ortalama idrar iyotu bizim çalışmamıza yakın olarak 107 µg/L olarak bulunmuştur. İdrar iyot düzeyi öğrencilerin 65'inde <20 µg/L, 185'inde 20-49 μg/L arası, 173'ünde 50-99 μg/L, 383'ünde 100-299 μg/L, 94 tanesinde ise >300 µg/L olarak sonuçlanmıştır [22].

Sonuç olarak Isparta ilinde okul çağı çocuklarında hafif düzeyde iyot eksikliği saptanmıştır. Çalışmamızda tespit edilen ortalama idrar iyot düzeyi bölgemizde yapılan daha önceki çalışmalarla karşılaştırıldığında İsparta ili okul çağı çocuklarının iyot alımının arttığı gösterilmiştir. Son 10-15 yıl içerisinde iyot profilaksisi programında önemli ilerleme sağlanmıştır ancak bölgede yapılan son taramanın üzerinden geçen 10 yıllık süreçte iyot eksikliğinin %72'den %63,7'ye gerilemesi yeterli olmayıp, bulunduğumuz coğrafyada iyot eksikliğinin hala devam ettiğini ve sürekliliğin sağlanması açısından profilaksinin devamı son derecede önemli olduğunu göstermektedir. Bu amaçla her 5 yılda bir bölgede idrar iyot miktarı ölçülmeli ve eksikliği durumunda önlemler alınmalıdır.

### Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı beyan edilmemiştir.

#### Kavnaklar

- 1. Misra S. Kantharia SL. Damor IR. Prevalence of goitre in 6-12 years schoolgoing children of Panchmahal district in Gujarat, India. Indian J Med Res 2007:126:475-9.
- 2. Zimmermann MB. Iodine deficiency. Endocr Rev 2009;30:376-408.
- 3. Bayram F, Beyazyıldız A, Gökçe C, Budak N, Erdoğan N, Kurtoğlu S et al. The prevalence of iodine deficiency, serum thyroglobulin, anti-thyroglobulin and thyroid peroxidase antibody levels in the urban areas of Kayseri, Central Anatolia. Exp Clin Endocrinol Diabetes 2009;117:64-8.
- 4. Aydın K, Kendirci M, Kurtoğlu S, Karaküçük El, Kiriş A. lodine and selenium deficiency in school-children in an endemic goiter area in Turkey. J Pediatr Endocrinol Metab 2002:15:1027-31.
- 5. DeLong GR, Leslie PW, Wang SH, Jiang XM, Zhang ML, Rakeman M et al. Effect on infant mortality of iodination of irrigation water in a severity iodine-deficient area of China. Lancet 1997;350:771-3.

- 6 Saniari M. Gholamhoseinian A. Nakhaee A. The Association between Cobalt Deficiency and Endemic Goiter in School-Aged Children. Endocrinol Metab (Seoul) 2014;29(3):307-11.
- 7. Assey VD, Peterson S, Kimboka S, Ngemera D, Mgoba C, Ruhiye DM et al. Tanzania national survey on iodine deficiency: impact after twelve years of salt iodation, BMC Public Health 2009:9:319.
- 8. Melse Bonstra A, Jaiswal N. lodine deficiency in pregnancy, infancy and childhood and its consequences for brain development. Best Pract Res Clin Endocrinol Metab 2010:24:29-38.
- 9. Gallego ML, Loango N, Londoño AL, Landazuri P. Urinary iodine excretion levels in schoolchildren from Quindío, 2006-2007. Rev Salud Publica (Bogota) 2009:11:952-60.
- 10. Zimmermann MB. The effects of iodine deficiency in pregnancy and infaancy. Pediatr Perinat Epidemiol 2012;26:108-17.
- 11. Eser S. Yurdumuzda goitre. İstanbul Ün Tıp Fak Mec 1956;19:114–8.
- 12. Hatemi H, Urgancıoğlu İ, Kaya H. Endemik Guatr Derecelendirilmesinde Bir İndeks Önerisi, Cerrahpaşa Guatr İndeksi. İstanbul Üniversitesi Cerrahpaşa Tıp Fakültesi Nükleer Tıp Anabilim Dalı Yavını: İstanbul. 1987.
- 13. Gür E. Ercan O. Can G. Akkus S. Güzelöz S. Ciftcili S et al. Prevalence and risk factors of iodine deficiency among schoolchildren. J Trop Pediatr 2003;49(3):168-
- 14. Kurtoglu S, Akcakus M, Kocaoglu C, Gunes T, Budak N, Atabek ME, et al. Iodine status remains critical in mother and infant in Central Anatolia (Kayseri) of Turkey. Eur J Nutr 2004;43(5):297-303.
- 15. Bastemir M, Emral R, Erdogan G, Gullu S.High prevalence of thyroid dysfunction and autoimmune thyroiditis in adolescents after elimination ofiodine deficiency in the Eastern Black Sea Region of Turkey. Thyroid 2006;16(12):1265-71.
- 16. Kutlu R, Karaköse S, Güngör K, Kulaksizoğlu S. The goiter prevalence and urinary iodine levels among adolescents. Turk J Pediatr 2011;53(2):161-8.
- 17. Budak N, Bayram F, Günay O, Kendirci M, Kurtoğlu S, Oz L. Iodine deficiency: an important and severe public health problem in Kayseri, Central Anatolia. J Endocrinol Invest 2007;30(11):920-4.
- 18. Cetin H, Kisioglu AN, Gursoy A, Bilaloglu E, Ayata A. Iodine deficiency and goiter prevalence in Turkey after mandatory iodization. J Endocrinol Invest 2006
- 19. Egri M, Ercan C, Karaoglu L.Iodine deficiency in pregnant women in eastern Turkey (Malatya Province): 7 years after the introduction of mandatory table salt iodization, Public Health Nutr 2009:12(6):849-52.
- 20. Ozkan B, Olgun H, Ceviz N, Polat P, Taysi S, Orbak Z et al. Assessment of goiter prevalence, iodine status and thyroid functions in school-age children of rural Yusufeli district in eastern Turkey. Turk J Pediatr 2004;46(1):16-21.
- 21. Akpınar D, Tanakol R, Boztepe H, Abbasoğlu S, Toker G, Alagöl F. İyotlama uygulama öncesi orta öğretim öğrencilerinde guatr sıklığı ve idrar iyot düzeyi. İstanbul Tip Fak 2002;65:176-81.
- 22. Erdoğan MF, Ağbaht K, Altunsu T, Ozbaş S, Yücesan F, Tezel B, Sargin C, Ilbeğ I, Artik N, Köse R, Erdoğan G. Current iodine status in Turkey. J Endocrinol Invest 2009;32(7):617-22.

#### How to cite this article:

Aslan N, Kurku H, Pirgon Ö. Urine Iodine Excretion and Iodine Deficiency Status in School Age Children at Isparta Province. J Clin Anal Med 2015;6(suppl 5): 612-6.



## **Pulmonary Atresia and** Ventricular Septal Defect in a Thirty-Six Year Old Woman

Pulmoner Atrezi ve Ventriküler Septal Defekt Birlikteliği / Pulmonary Atresia and Ventricular Septal Defect

İbrahim Koç<sup>1</sup>, Ayşen Dökme<sup>1</sup>, Nesimi Yavuz<sup>2</sup>, Gökhan Bulut<sup>3</sup> <sup>1</sup>Pulmonary Medicine, Viranşehir State Hospital, <sup>2</sup>Cardiology, Viranşehir State Hospital, <sup>3</sup>Radiology, Viranşehir State Hospital, Şanlıurfa, Turkey

Pulmoner atrezi ve ventriküler septal defekt (PA-VSD) konjenital kalp hastalığının nadir görülen bir formudur. Pulmoner arterlere kan akışı patent duktus arterious veya major aorto-pulmoner kollateral arterler (MAPCAs)'le sağlanır. Burada eforla artan nefes darlığı ve senkop nedeniyle kliniğimize refere edilen otuz altı yaşında Suriyeli savaş mağduru bir kadın hasta sunulmaktadır. Sorgulandığında çocukluğundan beri bu şikayetlerinin varlığını, kardiyak kateterizasyon ve ameliyat gerektiren bir hastalığının olduğunu ancak bu işlemleri kabul etmediğini belirtti. Ekokardiyografi ve bilgisayarlı tomografi anjiografide pulmoner atrezi ve ventriküler septal defet saptandı. Pulmoner arterlere kan akışının aortopulmoner kollateral arterlerce sağlandığı gözlendi. Pulmoner atrezili hastaların çoğunluğu ameliyat olmadan otuz yaşını geçememektedir. Burada sunulan olgu cerrahi işlem olmadan otuz yıldan fazla yaşayan nadir olgulardan biridir.

#### Anahtar Kelimeler

Pulmoner Atrezi; Ventriküler Septal Defekt; Senkop; Dispne

Pulmonary atresia with ventricular septal defect (PA-VSD) is a rare form of congenital heart disease. The blood supply to the pulmonary arteries is provided by a patent arterial duct or by major aorto-pulmonary collateral arteries (MAPCAs). Here we present a thirty six years old Syrian war victim who was referred to our clinic with complaints of dyspnea on exertion and syncope. When questioned she admitted that she was having these complaints since her childhood and had a disease that she was offered cardiac catheterization and operation which she refused. Echocardiographic and computed tomography angiography findings revelaed pulmonary atresia with ventricular septal defect and aortopulmonary collateral arteries provided the blood flow to pulmonary arteries. Most patients without surgery do not live more than three decades. The presented case is one of those surviving more than three decades without surgery.

#### Kevwords

Pulmonary Atresia; Ventricular Septal Defect; Syncope; Dyspnea

Corresponding Author: İbrahim Koç, Pulmonary Medicine, Viranşehir State Hospital, Şanlıurfa, Turkey. GSM: +905453142502 E-Mail: İbrahimkoc1981@gmail.com

#### Introduction

Pulmonary atresia with ventricular septal defect (PA-VSD) is a rare form of congenital heart disease. The blood supply to the pulmonary arteries is provided by a patent arterial duct or by major aortopulmonary collateral arteries (MAPCAs), which can vary greatly in number and in site of origin [1, 2]. Most of the cases with pulmonary atresia are associated with MAPCAs. It shares similarities with Tetralogy of Fallot (TOF) however, TOF involves pulmonary or infundibular stenosis but there is no pulmonary atresia. PA-VSD now is known as a distinct entity with its typical findings and management issues. The estimated survival rate without surgery is low [3]. PA with MAPCAs is a complex congenital cardiac anomaly and one of the most challenging groups to manage surgically. Echocardiography can be used to assess the presence and size of the central pulmonary arteries in patients with PA-VSD. Here we present a 36 years old woman Syrian war victim who had admitted to our clinic with dyspnea on exertion, low oxygen saturation and syncope and diagnosed with PA-VSD.

#### Case Report

A thirty six-year-old woman was admitted to our clinic with complaints of syncope, dyspnea on exertion and at rest. On her physical examination she had blurred consciousness, respiratory rate of 20 breaths per minute and SpO2 of 70% on room air, the patient had cyanotic and cluubbed fingers (Picture 1).



Picture 1. Cyanotic and clubbed fingers of the patient.

The patient was hospitalized for the further evaluation. Even under high oxygen supply SpO2 did not exceed 85%. When questioned she admitted that she was having these complaints since her childhood and had a disease that she was offered cardiac catheterization and operation which she refused. Computed tomography angiography of the chest revealed pulmonary artery atresia and aortopulmonary collateral arteries (Figure 1).





Figure 1. Computed tomography angiography of the chest revealed atresia of right and almost agenesis of left pulmonary artery and aortopulmonary collateral

Echocardiography revealed a large VSD which lay beneath the dilated aorta (Figure 2). Later on her follow up she admitted

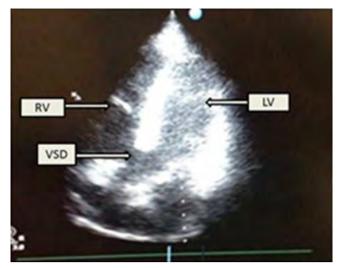


Figure 2. Echocardiography revealed a large VSD which lay beneath the dilated aorta, hypertrophied right ventricle.LV: Left ventricle, RV: Right ventricle, VSD: Ventricular septal defect.

that she had two pregnancies which resulted with abortion. In the light of her medical history, computed tomography angiography and echocardiography findings, she was diagnosed with pulmonary atresia and ventricular septal defect.

Pulmonary atresia with VSD is the ultimate form of TOF and is estimated to represent 5% to 10% of patients with Fallot tetralogy. Adult survivors of PA-VSD are quite rare: it is reported that the mean life expectancy without operation is not more than 3 decades [4]. Prognosis and survival of PA-VSD patients is dependent on the sufficiency of pulmonary blood flow derived from direct or indirect aortopulmonary collateral vessels. The well developed MAPCAs probably have enabled our patient to survive until this age. Especially left pulmonary atresia is almost agenesia but collaterals have provided the blood flow ( Figure 1). Echocardiography and multidetecter computed tomography are valuable non-invasive imaging modalities to evaluate VSD, the development and sources of MAPCAs in PA-VSD. Our case was a thirty- six years old woman who has been suffering from dyspnea on exertion and sometimes syncope. The patient refused to be operated and had two pragnencies which resulted with abortion. Main procedures are catheterization or surgical intervention. The reported surgical mortality has been relatively low and good functional results have been achieved [5, 6]. In the early stages of the life palliation is done by aortopulmonary shunt. The ultimate goal of surgery is to construct completely separated pulmonary and systemic circulations. This can be achieved as a single or staged procedure, depending on the pulmonary blood supply and complexity of the central pulmonary arteries. The surgical technique employed to repair PA-VSD defect with MAPCAs dependent pulmonary blood supply is variable, and depends upon individual anatomy and surgeon preference. Anatomic variability seen in PA-VSD makes the surgical approach patient specific [7]. Our case was offered surgery but she refused the procedure.

In conclusion patients with pulmonary atresia and ventricular septal defect rarely survive more than three decades, thanks to major aortopulmonary collateral arteries some patients manage to survive as in our patient.

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Rajeshkannan R, Moorthy S, Sreekumar KP, Ramachandran PV, Kumar RK, Remadevi KS. Role of 64-MDCT in evaluation of pulmonary atresia with ventricular septal defect. AJR Am J Roentgenol 2009;194(1):110-8.
- 2. Amin Z, McElhinney DB, Reddy VM, Moore P, Hanley FL, Teitel DF. Coronary to pulmonary artery collaterals in patients with pulmonary atresia and ventricular septal defect. Ann Thorac Surg 2000;70(1):119-23.
- 3. Lee CH, Kwak JG, Lee C, Primary repair of symptomatic neonates with tetralogy of Fallot with or without pulmonary atresia. Korean J Pediatr 2014;57(1):19-25.
- 4. Babliak OD, Lalyns'ka TA, Kurkevych AK, Maksymenko AV, Rudenko NM, Lemets IM. Pulmonary atresia with ventricular septal defect and major aorto-pulmonary collateral arteries: diagnosis and treatment. Lik Sprava 2015; 5(6): 94-7.
- 5. Atik, E. Case 3/2014 26-year-old man with pulmonary atresia and ventricular septal defect in late postoperative evolution. Arq Bras Cardiol 2014;102(4):34.
- 6. Perri G, Albanese SB, Carotti A. Airway complications after single-stage unifocalization for pulmonary atresia, ventricular septal defect, and major aortopulmonary collateral arteries. J Card Surg 2015;30(5):453-8.
- 7. Fouilloux V, Bonello B, Kammache I, Fraisse A, Mace L, Kreitmann B. Management of patients with pulmonary atresia, ventricular septal defect, hypoplastic pulmonary arteries and major aorto-pulmonary collaterals: Focus on the strategy of rehabilitation of the native pulmonary arteries. Arch Cardiovasc Dis 2012; 105(12):666-75.

#### How to cite this article:

Koç İ, Dökme A, Yavuz N, Bulut G. Pulmonary Atresia and Ventricular Septal Defect in a Thirty-Six Year Old Woman. J Clin Anal Med 2015;6(suppl 5): 617-9.

## Ductal Adenocarcinoma: A Rare Entity of Prostate Gland in a Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma Patient

## Kronik Lenfositik Lösemi/Küçük Hücreli Lenfositik Lenfomalı Bir Hastada Prostatın Nadir Bir Antitesi: Duktal Adenokarsinoma

KLL/SLL'li Bir Hastada Prostatin Duktal Adenokarsinoma / Prostatic Ductal Adenocarcinoma in a CII/SII Patient

Tumay Ozgur¹, Murat Mehmet Rifaioğlu², Ahmet Taner Sümbül³, Füsun Aydoğan⁴, Nesrin Atcı⁵¹Pathology Department, Mustafa Kemal University, School of Medicine, ²Urology Department, Mustafa Kemal University, School of Medicine, ³Oncology Department, Adana Başkent University, School of Medicine, ⁴Nuclear Medicine Department, Mustafa Kemal University, School of Medicine, ⁵Radiology Department, Mustafa Kemal University, School of Medicine, Hatay, Turkey

This case was presented as a poster in Avrasian Urooncology congress in 2015.

#### Özet

Prostat kanseri erkeklerde en sık gözlenen prostat malignensilerindendir ve duktal adenokarsinom klinik ve histolojik özellikleri ile belirgin patolojik bir subtipidir. Yetmiş altı yaşında Kronik lenfositik lösemi/Küçük hücreli lenfositik lenfomalı (KLL/SLL) erkek hasta alt üriner sistem semptomları ile hastanemize başvurdu. Son prostat spesifik antijen seviyesi (PSA) 26 ng/ml ile bir çok transrektal ultrasound rehberliğindeki biopsiler uygulandı ve patoloji raporları benign prostat hiperplazisi ve non-spesifik prostatit olarak sonuçlandı. Semptomların devam etmesi üzerine prostata transuretral rezeksiyon uygulandı. Materyalin patolojik değerlendirilmesinde benign hiperplazik prostat dokuları arasında stroması olmayan adenokarsinom odakları gözlendi. Tümör 4+4 Gleason patern skoru ile duktal adenokarsinom olarak tanı aldı. Metastaza bağlı lomber vertebrada kemik sintigrafisinde aktivite tutulumu vardı. Bilgisayarlı tomografide önceki KLL/SLL 'ye bağlı inguinal ve sağ iliak lenfadenopati izlendi. Total androjen kısıtlama tedavisi ve bilateral orşiektomi uygulandı. 3 ay sonra biokimyasal ve radyolojik görüntüleme sonuçlarına göre radyoterapi tedavisi planlandı. Duktal adenokarsinoma konvansiyonel adenokarsinomdan farklı klinik davranışı olan prostatın nadir bir subtipidir. Diğer yandan KLL/SLL takibi yapılan bir hastada sekonder bir malignensi olarak ortaya çıkması olguyu değerli kılmaktadır.

#### Anahtar Kelimeler

Duktal Adenokarsinoma; KLL/SLL; Prostat

#### Abstract

Prostate cancer is the most common malignancy in men and ductal adenocarcinoma is a pathologic subtype with specific histological and clinical features. Seventy-six year-old male patient with chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL) admitted to our hospital with lower urinary tract symptoms. The last prostate specific antigen (PSA) level was 26 ng/ml and serial transrectal ultrasound guided biopsies were administered and benign prostate hyperplasia and non-specific prostatitis were the results of pathology reports. Due to the persistence of the symptoms transurethral resection of the prostate was performed. In the pathologic evaluation of the material adenocarcinoma focuses without stroma has been observed between the hyperplasic prostate tissues. The tumor has been diagnosed as ductal adenocarcinoma with 4+4 Gleason pattern score. Bone scintigraphy was revealed activity uptake on lomber vertebral column due to metastasis. Computerized tomography was revealed previous bilateral inguinal and right iliac lymphadenopathy due to CLL/SLL. Total androgen deprivation therapy and bilateral orchiectomy was applied. After three mounts according to biochemical and imaging results, radiotherapy cure began. Ductal adenocarcinoma is a rare subtype of prostate carcinoma with clinical behavior from that seen in conventional adenocarcinoma. On the other hand it is worth to point out the occurence of this entity as second malignancy during follow-up of CLL/SLL.

#### Keywords

CLL/SLL; Ductal Adenocarcinoma; Prostate

DOI: 10.4328/JCAM.3560 Received: 11.05.2015 Accepted: 23.05.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 620-2 Corresponding Author: Tumay Ozgur, Pathology Department, Mustafa Kemal University, School of Medicine, 31100, Serinyol, Hatay, Turkey. T.: +90 3262291000-3329 F.: +90 3262455654 E-Mail: ozgurtumay@yahoo.com

#### Introduction

Ductal adenocarcinoma (DA) of the prostate is a rare histologic subtype of prostate cancer that was first defined by Melicow et al. as endometrial carcinoma of prostatic utricule in 1967 [1]. Prostate spesific antigen levels (PSA) seem to be in normal levels and the behaviour of the tumor is uncertain [2].

DA usually presents with aciner adenocarcinoma and the pure form is rare constituting 0.4-0.8 % of all radical prostatectomy and biopsy specimens [3]. It may be misdiagnosed as various benign, precancerous and malign lesions like prostatic urethral polyps, high grade prostatic intraepithelial neoplasia (HG-PIN) and colorectal adenocarcinoma.

Postatic DA may originate either from large primary periurethral prostatic ducts or rarely from peripheral prostatic ducts [4].

We would like to describe our case which we diagnosed in transurethral resection specimen incidentally as DA with mild PSA levels and prostatism symptoms in a chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL) patient.

#### Case Report

Seventy-six year-old male patient with CLL/SLL admitted to Mustafa Kemal University hospital Urology department outpatient clinic with lower urinary tract symptoms. Prostate gland was 90 gram in ultrasonography (US) examination. The last total PSA and free PSA level was 23.3 and 5.7 ng/ml, respectively and three consecutive twelve core transrectal ultrasound guided biopsies were administered and benign prostate hyperplasia and non-specific prostatitis were the results of pathology reports. Due to the persistence of the symptoms transurethral resection of the prostate (TURP) was performed, during TURP procedure there was a papillary lesion protruded from right apex of the prostatic urethra and the material has been sent to pathology laboratory. In the microscopic evaluation of the specimen adenocarcinoma focuses without stroma has been observed between the hyperplasic prostate tissues (Figure 1). The nuclear morphology was different from classic acinar adenocarcinoma. To confirm the diagnosis and differentiate it from metastatic tumors an immunohistochemical panel was applied. While CK7, CK20 were negative, PSA and AMACR were positive in immunohistochemical studies (Figure 2-3). The tumor has been diagnosed as ductal adenocarcinoma with 4+4 Gleason pattern score. Bone scintigraphy was revealed activity uptake on lomber vertebral column. Computerized tomography was revealed previous bilateral inguinal and right iliac lymphadenopathy due to CLL. Total androgen deprivation therapy and orchiectomy was applied, after three mounts according to biochemical and imaging results, radiotherapy cure was began.

#### **Discussion**

Prostatic DA is a rare variant most commonly growing as exophytic mass lesions in the uretra. There are various mimickers of this entity that it should be recognised by pathologists and uropathologists in daily practice [4].

DAs are often localised at the central ducts of the gland. Therefore they are usually seen in TURP and radical prostatectomy specimens and less diagnosed in needle biopsies. Our case had also two serial transrectal US guided biopsies but final proce-

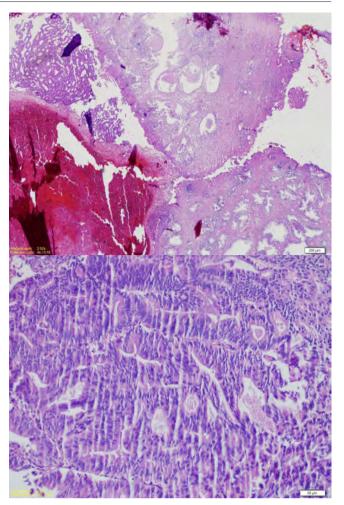


Figure 1-2. Adenoid and cribriform structures with atypical columnar epithelium without stroma between the benign prostate glands (Hematoxilen&Eosinx200).

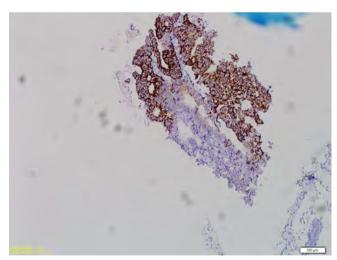


Figure 3. AMACR positivity in tumor cells (AMACRX100).

dure with TURP demonstrated the tumor. Sfoungaristos et al. reported an 82 year old Caucasian man with DA that was diagnosed in TURP specimen like our case [5].

DA is seen in elderly man with a range of 65 to 87 with obstructive symptoms and gross hematuria [6]. Our patient was 76 years old at the time of diagnosis and admitted to our clinic with lower urinary tract symptoms.

DA cells express PSA but PSA levels are not always high in these tumors as in our patient (26 ng/ml) that could be in inflammation or hyperplasias of the gland.

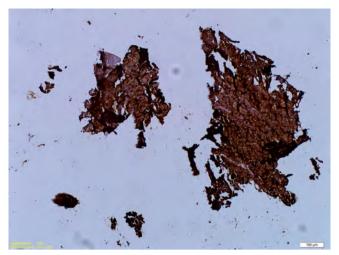


Figure 4. PSA positivity in tumor cells (PSAX100).

The cystourethroscopic appearence of DA is described as exophytic and polypoid mass projecting into the uretra or near the verumontanum [6]. Kan et al. defined 10 of their cases as intra luminal growth masses during cystoscopy in their review of Hong Kong DA series [2]. We have observed an exophytic mass lesion projecting into the lumen at the prostate apex with right wall laterilization.

DA has benign and malign mimickers. Prostatic urethral polyp is a polipoid lesion lined by benign appearing prostatic glandular epithelium and it can be confused with DA in fragmentated needle biopsies. HG-PIN cribriform type is a difficult entitity that should be remembered in differential diagnosis. HG-PIN constitutes with micropapillary cores while DA presents with true papillary cores. Beside this DA is recognisable with distinct atypia and large, back to back glands and usually comedonecrosis. DA might be confused with colorectal carcinoma metastasis. The verification of the diagnosis need immunohistochemical studies like PSA and PSAP. Another tumoral lesion that we should remember is papillary urothelial carcinoma in differential diagnosis; the cribriform architecture of DA is missing and nuclei of the tumor cells are more pleomorphic [4]. In the microscobic evaluation of the specimen we have found tumor composed of cribriform and adenoid structures with atypical columnar cells between the hyperplastic prostatic tissues. We have immunostained the tumoral tissue by Cytokeratin 7 and 20, AMACR, P63 and PSA. While Cytokeratin 7, 20 were negative, AMACR and PSA were strong and diffuse positive confirming our diagnosis of prostatic DA. P63 was diminished in neoplastic ducts.

In the literature DA is defined as a more agressive neoplasm compared to aciner adenocarcinoma. DA is mostly graded as 4+4= 8 whereas in cases of mixed ductal and aciner patterns ductal component should be assigned to Gleason patern 4 according to Gleason histopathologic scoring system [2]. We have also reported our case as 4+4=8 with its morphologic architecture and ductal differentiation.

Kan et al. reported bone and rectum invasions in 7 of their 20 DAs [2]. Our patient has lomber vertebra metastasis and alive receiving radiotherapy with no morbidity.

Engin et al. declared the incidence of multipl primary cancers as 0.83% among cancer patients [7]. CLL/SLL patients are candidates for developing a second cancer and an increased frequency of certain cancer types twice times more compared

to normal population because of disease or therapy related immunsupression. Tsimberidau et al. reported prostate carcinoma synchronous with CLL in 12.8 % of their series [8]. Our patient is also under follow-up for CLL/SLL for three years and developed DA after two years.

DA of the prostate has unique morphological, clinical features and agressive behaviour. It should be remembered in cases with non-specific pathologic findings in periferic prostate biopsies of patients with persisant lower urinary tract symptoms. We should also point out this rare entity with its occurence during CLL/SLL follow-up.

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Melicow MM, Pachter MR. Endometrial carcinoma of prostatic utricle (uterus masculinus). Cancer 1967;20(10):1715-22.
- 2. Kan RWM, Kan CF, Wong JHM, Fu KK, Ng CF, Chan SW. Ductal adenocarcinoma of the prostate: a Hong Kong case series. Int Urol Nephrol 2014:46:2133-37.
- 3. Greene LF, Farrow GM, Ravits JM, Tomera FM. Prostatic adenocarcinoma of ductal origin. J Urol 1979:121:303-5.
- 4. Epstein Jl. Prostatic ductal adenocarcinoma: A Mini Review. Medl Princ Pract 2010:19:82-5
- 5. Sfoungaristos S, Katafigiotis IS, Tyritzis S, Kavouras A, Kanatas P, Petas A. An 82-year-old Caucasian man with a ductal prostate adenocarcinoma with unusual cystoscopic appearance: a case report. J Med Case Rep 2011; 5:4.
- 6. Brinker DA, Potter SR, Epstein JI. Ductal adenocarcinoma of the prostate diagnosed on needle biopsy: correlation with clinical and radical prostatectomy findings and progression. Am I Surg Pathol 1999.23:1471-9.
- 7. Engin K. Cancers in multipl primary sites. Int Surg 1994;79:33-7.
- 8. Tsimberidou AM, Wen S, McLaughlin P, et al. Other malignancies in chronic lymphocytic leukemia/small lymphocytic lymphoma. J Clin Oncol 2009; 27:904-10.

#### How to cite this article:

Ozgur T, Rifaioğlu MM, Sünbül AT, Aydoğan F, Atcı N. Ductal Adenocarcinoma: A Rare Entity of Prostate Gland in a Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma Patient. J Clin Anal Med 2015;6(suppl 5): 620-2.



## **Amniotic Band Syndrome and a Rarely Seen Subtype: Limb Body Wall Complex**

Amniyotik Bant Sendromu Subtipi: Vücut Ekstremite Duvar Defekti / Amniotic Band Syndrome Subtype: Limb Body Wall Complex

Derya Akdağ Cırık, Mehmet Metin Altay, Metin Kaplan, Füsun Bocutoğlu, Orhan Gelisen Department of Obstetrics and Gynecology, Etlik Zubeyde Hanım Women's Health Training and Research Hospital, Ankara, Turkey

Amniyotik Bant Sendromu, fibröz amniyotik bantların fetal dokuları sıkıştırması ile ortaya çıktığı düşünülen çok çeşitli fetal konjenital anomalileri kapsayan nadir bir konjenital bozukluktur. Hastalardaki semptomlar minör parmak ampütasyonlarından fatal konjenital anomalilere kadar uzanan geniş bir spektrumda izlenir. Erken ikinci trimesterde prenatal ultrasonografide belirli ultrasonografik bulguları ile amniyotik bant sendromunun ciddiyeti saptanabilir. Amniyotik bant sendromunun fatal bir subtipi olan ekstremite vücut duvar kompleksinin erken dönemde tanınması hastaların bilgilendirilmesi ve erken dönemde terminasyonuna olanak sağlar. Biz burada erken ikinci trimesterde tanısını koyduğumuz ve termine ettiğimiz vücut ekstremite duvar kompleksine sahip bir olguyu sunmak ve prenatal ultrasonografik bulgularını tartışmayı amaçladık.

Amniyotik Bant Sendromu; Fetal Anomali; Prenatal Ultrasonografi

Amniotic band syndrome is a rare congenital disorder that is caused by intrauterine entrapment of fetal parts in fibrous amniotic bands. Patients may have varying clinical features ranging from minor digital amputations to fatal congenital anomalies. Prenatal ultrasound scan regarding the specific findings of amniotik bant sendromu during the early second trimester may provide the detection of the severity of the disease. Because Limb Body Wall Complex is a lethal condition, earlier diagnosis may also provide to counsel the parents and terminate the pregnancy in an earlier period. We herein present a case of Limb Body Wall Complex, diagnosed and terminated at 14 weeks of gestation and discuss the ultrasonographic findings.

Amniotic Band Syndrome; Fetal Anomaly; Prenatal Ultrasonography

DOI: 10.4328/JCAM.3593 Corresponding Author: Derya Akdağ Cırık, Yeni Etlik Caddesi No: 55 Etlik, Kecioren, Ankara, Türkiye. GSM: +905066288930 F.: +90 3123220181 E-Mail: deryaakdag@yahoo.com

#### Introduction

Amniotic band syndrome (ABS) is a rare congenital disorder, thought to develop secondary to the compression of fetal tissues by fibrous amniotic bands. Its incidence is one out of 1,200 to 15,000 live births [1]. It is a sporadic condition and does not have genetic origin. Two main pathogenesis has been introduced to explain the pathogenesis of this disease; one is the exogenous theory that proposed the early rupture of the amniotic membrane leading to fibrous bands. These fibrous bands usually entrap a part of extremity, reduce the blood circulation and lead to autoamputation of a digit or entire limb in utero [2]. The other endogenous theory proposes the vascular problems as a cause of the condition [3]. Although the most common finding of this syndrome is digital amputation, it may not be seen in all cases [4]. Other observed anomalies are as follows; contraction rings, lymphedema, craniofacial defects, encephalocele, anencephaly, drop foot and multiple joint contractures [5]. Although the diagnosis is possible in ABS by prenatal ultrasonography, the fetuses are usually diagnosed by postpartum physical examination [6]. We aimed to present a case of a Limb Body Wall Complex (LBWC), the fatal subtype of ABS, which was diagnosed during the early second trimester (14th weeks of gestation) ultrasound scan.

#### Case Report

A 31-year-old G1 case was referred to our early pregnancy clinic with the suspicion of multiple fetal abnormalities. Her medical history revealed no prior systemic diseases and drug use, no history of infection or trauma during pregnancy, and no consanguineous marriage. The ultrasonography examination revealed amniotic fluid in normal ranges, and the presence of a live fetus with a CRL of 80 mm, compatible with 14 weeks. Fetal movements were free at the lower extremities, but the upper extremity and the head were noted not to move extensively. The detailed ultrasonography examination revealed wrapping amniotic bands, especially the left hand and around the head of the fetus, meandering as loose fibers in the amniotic cavity. The upper right extremity of the fetus was seen to be totally absent starting from the shoulder level. Both lower extremities on the other hand were observed to be in normal dimensions and moving freely. The fetal cranium was seen to be totally absent above the bilateral orbita at the anterior and occipital bone at the posterior site and the brain tissue was seen to be free in the amniotic fluid (Figure 1). An abdominal wall defect at the



Figure 1. Ultrasound scan performed at 14 gestational weeks. Left arrow points at the fetal exencephaly. Right arrow points the left hand in a fixed extended position due to entrapment of the amniotic bands.

umbilicus in the fetal anterior wall and fetal intestines expulsed out to the amniotic cavity through this defect were observed (Figure 2). The diagnosis with those findings was 'LBWC' and

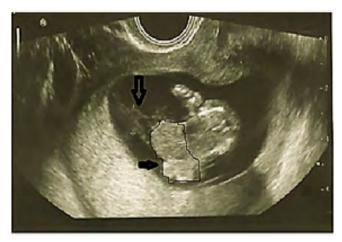


Figure 2. Ultrasound scan performed at 14 gestational weeks. Thin arrow points the protrusion of the fetal abdominal organs through the thoraco-abdominal wall defect. Thick arrow points the attachment between these structures and fetal

detailed information was given to the patient about the condition of the fetus and possible perinatal results of the condition. Upon the request by the patient to terminate the pregnancy, she signed for approval and a medical termination protocol was applied. Upon morphological examination of the fetus after the abortus, distal phalangeal amputations were observed in the left hand on the fourth and fifth fingers, while the right upper extremity was totally absent (Figure 3). A cranial defect starting

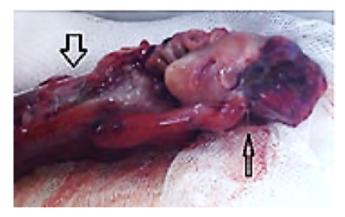


Figure 3. A photograph of fetus taken after abortion. Thin arrow points the amniotic band around the fetal left hand and absence of fetal cranium above the level of orbita. Thick arrow points the wide asymmetrical thoraco-abdominal wall

from the bilateral fetal orbita to the occipital bone and a disoriented cortex not covered with calvarium and an anterior wall defect starting from the anterior fetal abdominal wall extending to the left axilla including the thoracic wall. The intestines were observed to be expulsed through this defect and they were seen outside the abdomen (Figure 4).

#### **Discussion**

The case reported herein is compatible with the diagnosis of LBWC, which is a fatal subtype of amniotic band syndrome. The diagnosis of LBWC requires at least two of the following three criteria, which are exencephaly and facial clefts, thoracic and/or abdominoschisis, and extremity defects [7]. The case presented herein meets all three criteria. In addition to the upper extrem-

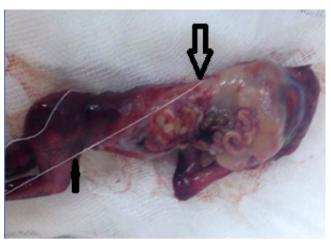


Figure 4. A photograph of fetus taken after abortion. Thick arrow points the absence of left upper extremity and expulsion of fetal intestines through the fetal body wall defect. Thin arrow points the white fetal amniotic bands surrounding the fetal body.

ity abnormalities in this case, both a craniofacial defect and an atypical wall defect including both the thorax and abdomen were present. Internal organ defects are generally present in 95% of the cases of LBWC and more than 70% of those defects are thought to develop secondary to vascular disorders [8]. However, since the case presented herein was diagnosed at an earlier stage of pregnancy and since no postmortem pathological study was performed, information about the internal organ defects is not provided.

Other non-lethal conditions that might be confused with LBWC are polymalformation complexes such as Cantrell Pentalogy, omphalocele-exstrophy-imperforated anus-spinal defects, and isolated gastroschisis [9]. Five typical findings necessary to be present for the diagnosis of Cantrell Pentalogy are cardiac abnormalities such as ectopic cordis, lower sternal defects, midline supraumbilical wall defects, anterior diaphragmatic defects, and defects of the diaphragmatic segment of the pericardium. On the other hand, ultrasonography findings of omphaloceleexostrophy-imperforated anus-spinal defects complex are absence of fetal bladder, presence of an infraumbilical abdominal wall defect, spinal distortion, and a sacral myelomeningocele [10]. In contrast to LBWC, the fetal intestines are covered with peritoneum in the two syndromes stated above. The fetal intestines are freely present in the amniotic cavity in gastroschisis; however, there is no additional wall defect.

Since LBWC is a lethal condition, early diagnosis of this syndrome provides an opportunity to terminate the pregnancy in an earlier period. Therefore it should be differentiated from other syndromes with a careful prenatal ultrasonography as early as possible. Therefore, obstetricians should carefully examine the fetus regarding these specific ultrasonographic findings during the early second trimester in order to counsel the parents with an early diagnosis of LBWC.

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Garza A, Cordero JF, Mulinare J. Epidemiology of the early amnion rupture spectrum and defects. Am J Dis Child 1988;142(5):541-4.
- 2. Tadmor OP, Kreisberg GA, Achiron R, Porat S, Yagel S. Limb amputation in amniotic band syndrome: serial ultrasonographic and Doppler observations. Ultrasound

- Obstet Gynecol 1997:10(5):312-5
- 3. Sentilhes L, Verspyck E, Patrier S, Eurin D, Lechevallier J, Marpeau L. Amniotic band syndrome: pathogenesis, prenatal diagnosis and neonatal management. J Gynecol Obstet Biol Reprod (Paris). 2003;32(8):693-704
- 4. Marino T. Ultrasound abnormalities of the amniotic fluid, membranes, umblical cord and plasenta. Obstet Gynecol Clin North Am 2004;31(1):117-200.
- 5. Goldfarb CA, Sathienkijkanchai A, Robin NH. Amniotic constriction band: a multidisciplinary assessment of etiology and clinical presentation. I Bone Joint Surg Am 2009:91(4):68-75.
- 6. Aynioglu O, Erol AS, Baser R, Isik H, Sahbaz A. Amniotic Band Syndrome in a Pregnancy with Uterin Anomaly, A Case Report. J Clin Anal Med 2013; DOI: 10.4328/ JCAM.2180
- 7. Van Allen MI, Curry C, Gallagher L. Limb body wall complex: I. Pathogenesis. Am J Med Genet 1987;28(3):529-48
- 8. Pumberger W, Schaller A, Bernaschek G. Limb-body wall complex: A compound anomaly pattern in body-wall defects. Pediatr Surg Int 2001;17(5-6):486-90.
- 9. Kocherla K, Kumari V, Kocherla PR. Prenatal diagnosis of body stalk complex: A rare entity and review of literature, Indian I Radiol Imaging 2015;25(1):67-70. 10. Keppler-Noreuil K, Gorton S, Foo F, Yankowitz J, Keegan C. Prenatal ascertainment of OEIS complex/cloacal exstrophy -  $15\,\mathrm{new}$  cases and literature review. Am J Med Genet 2007;143(18):2122-8.

#### How to cite this article:

Cırık DA, Altay MM, Kaplan M, Bocutoğlu F, Gelisen O. Amniotic Band Syndrome and a Rarely Seen Subtype: Limb Body Wall Complex. J Clin Anal Med 2015;6(suppl



## Treatment of Vaginal Agglutination Complicating Chronic Graft-Versus-Host Disease: A Rare Case Report

## Kronik Graft-Versus-Host Hastalığında Vajinal Aglütinasyon Tedavisi: Olgu Sunumu

Kronik Graft-Versus-Host Hastalığı / Chronic Graft-Versus-Host Disease

Suleyman Engin Akhan<sup>1</sup>, Hande Delier<sup>1</sup>, Ercan Bastu<sup>1</sup>, Cenk Yasa<sup>1</sup>, Funda Gungor Ugurlucan<sup>1</sup>, Sevgi Kalayoglu-Besisik<sup>2</sup>
<sup>1</sup>Department of Obstetrics and Gynecology, <sup>2</sup>Division of Bone Marrow Transplantation, Department of Internal Medicine,

Istanbul University School of Medicine, Istanbul, Turkey

#### Özet

Myelodisplastik sendrom tedavisi için iki yıl önce kemik iliği nakli olan 34 yaşındaki hasta vajinal aglütinasyona bağlı cinsel birlikteliği girememe şikayeti ile jinekooji kliniğimze başvurdu. Vajinal adezyonlar cerrahi yaklaşımla tedavi edildikten sonra topikal estrojen ve tibolon tedavileri başlandı. Ameliyattan iki ay sonra hasta cinsel ilişkiye girebildi. Bu olgu sunumunda graft versus host hastalığının vulvovajinal komplikasyonları, tedavisi ve önlenmesi tartışılmaktadır.

#### Anahtar Kelimeler

Kemik İliği Nakli; Graft-Versus-Host Hastalığı; Vajinal Aglütinasyon

#### Abstract

We describe a 34-year old patient with vaginal agglutination, which presented as inability to have sexual intercourse two years after bone marrow transplantation for myelodysplastic syndrome. The vaginal adhesions were freed surgically after local estrogen therapy and tibolone was started. She was able to have sexual intercourse two months after the operation. In this case report, vulvovaginal complications of graft-versus-host disease and their treatment and prevention are discussed.

#### Keywords

Bone Marrow Transplantation; Graft-Versus-Host Disease; Vaginal Agglutination

DOI: 10.4328/JCAM.3592 Received: 13.05.2015 Accepted: 02.06.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 626-8 Corresponding Author: Ercan Bastu, Department of Obstetrics and Gynecology, Istanbul University School of Medicine, 34093 Capa, Istanbul, Turkey. GSM: +905324134195 E-Mail: ercan.bastu@istanbul.edu.tr

#### Introduction

Allogenic blood or bone marrow transplantation (BMT) has become an attractive treatment alternative for various diseases, particularly for hematologic diseases. Graft-versus-host-disease (GVHD), an immunologic disorder in which immunocompetent donor T lymphocytes recognize, become sensitized to and react against histocompatibility antigens of the host is a major complication of allogenic BMT. Chronic GVHD affects approximately 25-45% of patients and occurs in >100 days after BMT. Both limited (only skin or liver) and extensive (skin, liver, oral mucosa, lacrimal glands, esophagus, and serosal membranes) forms of chronic GVHD resemble collagen vascular diseases [1]. Vulvovaginal complications such as vulvar atrophy, alopecia of the vulva, vaginal synechiae, stricture and complete obliteration leading to secondary complications such as hematocolpometra may also develop in female patients with chronic GVHD [2-3]. Here, we describe a patient who received allogenic peripheral blood stem cell transplantation (PBSCT) for myelodysplastic syndrome (MDS) and developed chronic GVHD leading to vaginal agglutination.

#### Case Report

A 34 year-old, gravida 2, para 2 woman with MDS underwent allogenic PBSCT in January 2012 after conditioned with busulfan and cyclophosphamide. GVHD prophylaxis consisted of cyclosporine and short-term methotrexate. On the 20th day, she developed acute GVHD with grade II skin involvement that required high dose methylprednisolone treatment. She gradually progressed to extensive chronic GVHD with skin and oral mucosa involvement. Sicca syndrome (Sjögren's syndrome) accompanied the clinical course. Extracorporeal phototherapy was initiated to reduce steroid usage, which led to serious osteoporosis with compression fractures despite prophylaxis with calcium and vitamin D.

At the end of the second year after the transplant, she came to our clinic with a complaint of inability to have sexual intercourse. She also developed secondary amenorrhea since the transplantation. Gynecologic examination revealed vulvar atrophy, alopecia of the vulva and pubis, and complete obliteration of the introitus. The uterus was atrophic, endometrial thickness was 4 mm, and both of the ovaries were also atrophic and nonfollicular according to transrectal sonographic examination. Follicle-stimulating hormone and luteinizing-hormone (LH) levels were elevated, while estradiol (E2) level was low; 132.1mIU/ ml, 57.1mlU/ml, and 5.0pg/ml, respectively. After a month of local estrogen (estriol 1mg/g) usage, dense vaginal adhesions obliterating the lower half of the vagina were freed with blunt and sharp dissection up to the level where the original vaginal mucosa and cervix could be visualized under general anesthesia. The vaginal mucosa and ectocervix were atrophic and fragile. A 3x8cm vaginal dilator made of glass was placed into the vagina and left in place for two days. Tibolone (2.5mg/day, PO) was also administered. The patient continued to use the vaginal dilator and estrogen cream, and returned for pelvic examination three weeks postoperatively, which revealed normal vaginal patency. She was advised to have regular intercourse and to use the mold only during nights. At the two-month postoperative visit, the patient reported that she was able to have comfort-

able sexual intercourse. Therefore, she was advised to cease the vaginal dilator usage.

#### Discussion

In 1982, Corson et al. have first addressed gynecologic manifestations of chronic GVHD [2]. They have described four patients with chronic GVHD who developed vaginal inflammation, sicca syndrome, adhesions or stenosis requiring surgical therapy. Vaginal agglutination and its potential complications such as hematocolpometra and endometriosis, related to chronic GVHD have also been reported in six other case reports [3-8]. Our case, to our knowledge, is one of the very few cases of vaginal agglutination accompanying chronic GVHD described in the literature. Secondary amenorrhea either due to vaginal agglutination or ovarian failure due to chemo-irradiation damage often accompanies vulvovaginal problems in these patients. Our patient also had secondary amenorrhea, premature ovarian failure and dense vaginal adhesions.

The etiology of gynecologic problems in allogenic PBSCT patients is multifactorial [7]. One of the causes of premature ovarian failure in this context is the toxicity of the conditioning regimens in myeloablative doses. Abnormalities of humoral and cellular immunity associated with chronic GVHD resembling autoimmune diseases may also contribute to the ovarian failure [1]. However, the histopathologic effects of chronic GVHD itself may explain vulvar atrophy, vaginal dryness, adhesions and strictures in our patient. Hypoestrogenism as a result of concomitant ovarian failure seemed to exacerbate the vulvovaginal symptoms.

Young patients with GVHD usually have iatrogenic premature ovarian failure as well. Therefore, their treatment should include every effort to raise the quality of life, including hormone treatment. In the first case series of 5 patients with gynecologic manifestations of GVHD reported by Corson et al., it has been emphasized that therapy of vaginal manifestations of chronic GVHD should be both local and systemic [2]. We preferred tibolone, which had androgenic activity for our patient, believing that it would have a positive effect both on her mood and libido. However, the preferred type of hormone therapy for these patients needs further investigation. In another case series of 8 patients, Constantini et al. presented the importance of compliance to vaginal dilator program [9], which is in line with our case. As, Haemato-oncology subgroup of the British Committee for Standards in Haematology (BCSH) and the British Society for Bone Marrow Transplantation (BSBMT) have concluded in a joint working group the ultimate goal of the treatment, whichever one is chosen, should be the effective control of GVHD while minimizing the risk of toxicity and relapse [10].

#### Conclusion

Gynecologic problems may be underestimated during the late period of PBSCT especially in patients with chronic GVHD. Therefore, regular gynecologic examination may prevent complications such as vaginal agglutination, which can result in hematocolpometra, endometriosis and low quality of life due to inability to have sexual intercourse. Early diagnosis is very important since simple measurements such as local estrogen creams and vaginal dilators may be helpful. On the other hand,

for established vaginal strictures, surgery is required, which has the risk of inadvertent bladder and rectal damage due to atrophy and fibrosis.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Sullivan KM, Shulman HM, Storb R, Weiden PL, Witherspoon RP, McDonald GB, et al. Chronic graft-versus-host disease in fifty-two patients: Adverse natural course and successful treatment with combination immunosuppression. Blood 1981:57:267.
- 2. Corson SL, Sullivan K, Batzer F, August C, Storb R, Thomas ED. Gynecologic manifestations of chronic graft-versus-host disease. Obstet Gynecol 1982;60:488-92.
- 3. Anguenot J, Ibéchéole V, Helg C, Piacenza J, Dumps P, Bonnefoi H. Vaginal stenosis with hematocolpometra, complicating chronic graft versus host disease. Eur J Obstet Gynecol Reprod Biol 2002;103(2):185-7.
- 4. Hayes EC, Rock JA. Treatment of vaginal agglutination associated with chronic graft-versus-host disease. Fertil Steril 2002;78(5):1125-6.
- 5. Yanai N, Shufaro Y, Or R, Meirow D. Vaginal outflow tract obstruction by graftversus-host reaction. Bone Marrow Transplant 1999;24(7):811-2.
- 6. Louis-Sylvestre C, Haddad B, Paniel BJ. Treatment of vaginal outflow tract obstruction in graft-versus-host reaction. Am J Obstet Gynecol 2003;188(4):943-4.
- 7. De Lord C, Treleaven J, Shepherd J, Saso R, Powles RL. Vaginal stenosis following allogeneic bone marrow transplantation for acute myeloid leukaemia. Bone Marrow Transplant 1999;23(5):523-5.
- 8. Hundley AF, Magro C, Bartholomew D. Vaginal agglutination: chronic graftversus-host disease mimics lichen planus. J Low Genit Tract Dis 2011;15(1):71-4. 9. Costantini S, Di Capua E, Bosi S, Chiodi S, Spinelli S. The management of severe vaginal obstruction from genital chronic graft-versus-host disease: diagnosis, surgical technique and follow-up. Minerva Ginecologica 2006;58:11-6.
- 10. Dignan FL, Amrolia P, Clark A, Cornish J, Jackson G, Mahendra P, et al. Diagnosis and management of chronic graft-versus-host disease. Br J Haematol 2012;158(1):46-61.

#### How to cite this article:

Akhan SE, Delier H, Bastu E, Yasa C, Ugurlucan FG, Kalayoglu-Besisik S. Treatment Of Vaginal Agglutination Complicating Chronic Graft-Versus-Host Disease: A Rare Case Report. J Clin Anal Med 2015;6(suppl 5): 626-8.



## SCIWORA at Thoracic Level in an Adult: A Case Report

## Erişkinde Torakal Seviyede SCIWORA: Olgu Sunumu

Erişkinde SCIWORA / SCIWORA in an Adult

Seniz Akcay Yalbuzdag<sup>1</sup>, Taciser Kaya<sup>1</sup>, Ilker Sengul<sup>1</sup>, Altinay Goksel Karatepe<sup>1</sup>, Zehra Hilal Adibelli<sup>2</sup> <sup>1</sup>Physical Medicine and Rehabilitation, <sup>2</sup>Radiology, Izmir Bozyaka Education and Research Hospital, Izmir, Turkey

Presented as case report-Poster at 25. National Physical Medicine and Rehabilitation, 22-26 April 2015, Antalya, Turkey

SCIWORA daha sıklıkla çocuklarda tanımlanmış olmasına rağmen, az sayıda yayında erişkinde SCIWORA olguları bildirilmiştir. 27 yaşında erkek hasta, kliniğimize 8 metre yüksekten düşme ve eşzamanlı torakal bölgeye olan künt travma sonrası gelişen parapleji tablosu ile sevkedildi. Hastanın nörolojik seviyesi , Amerikan Spinal Yaralanma Bozukluk skalası' na göre T11 (AIS) C olarak belirlendi. Servikal, torakal, lomber ve beyin manyetik rezonans görüntülemeleri (MRG) T8-9, T9-10 intervertebral disk protrüzyonu dışında herhangi bir spinal kord veya ligamentöz yaralanma ile uyumlu değildi. Ürodinamik çalışma sonuçları flask nörojenik mesane ile uyumluydu. Hasta nörolojik rehabilitasyon programına alındı. Nörolojik rehabilitasyon programından sonra hastanın fonksiyonel durumunda parsiyel iyileşme gözlendi. MRG' de spinal kord anormalliği saptanmadığı durumlarda SCIWORA tanısını koymak zor olabilir. Bu yüzden tanı spinal şokun sonlanmasına kadar gecikebilir.

#### Anahtar Kelimeler

SCIWORA; Spinal Kord Yaralanması; Erişkin

#### Abstract

Although SCIWORA has been reported more prevalent in children, few case reports published SCIWORA in adults. Twenty seven year old man transferred to our rehabilitation unit with paraplegia arose after fall from height of 8 and concurrent blunt trauma to thoracic region. The neurological level was of T11 American Spinal Injury Association Impairment Scale (AIS) C. Magnetic resonance imaging (MRI) findings of both brain, and spine didn't prove any significant abnormalities except the protrusion of intervertebral discs at the levels of T8-9, and T9-10. Urodynamic study findings were compatible with flaccid neurogenic bladder. He enrolled in the neurological rehabilitation program. After neurological rehabilitation program partial improvement observed in his functional status. The diagnosis of SCIWORA without any abnormality in the spine with MRI may be challenging. Therefore the diagnosis might retard until the termination of spinal shock.

### Kevwords

SCIWORA; Spinal Cord Injury; Adult

DOI: 10.4328/JCAM.3583 Corresponding Author: Seniz Akcay Yalbuzdag, Physical Medicine and Rehabilitation, Izmir Bozyaka Education and Research Hospital, Izmir, Turkey. T.: +90 2322505050 GSM: +905333798669 E-Mail: senizakcay@hotmail.com

SCIWORA has been described as spinal cord injury without any abnormalities in both radiography, and computed tomography [1]. Availability and increasing usage magnetic resonance imaging (MRI), the diagnosis of SCIWORA resulted in differences in the diagnosis of SCIWORA, with regard to definition, and classification. The term 'spinal cord injury without radiologic evidence of trauma' (SCIWORET) was recommended recently for adults who has no evidence of trauma on plain radiographs, and computed tomography [2]

The spinal cord is more vulnerable to injury without radiographic abnormality in children by comparison with adults and it affects mostly the cervical spine [1]. Hereby we present an atypical case of adult SCIWORA in the thoracic region without any abnormality identified at the spinal cord detected by MRI.

### Case Report

Twenty seven year old male was transferred to our tertiary inpatient rehabilitation unit with paraplegia, which has developed after falling from a height of 8 meters and concurrent blunt trauma to thoracic region. He described weakness, numbness in lower extremities accompanying with urinary and bowel incontinence. The initial manual muscle testing revealed that, the hip flexors, and knee extensors were of grade 1/5, and other muscle groups were of grade 0/5 motor function at lower extremities. Dermatomas under T-11 were all anesthethic. He had no relevant disease history except the L4-5 disc herniation operation 5 months ago. Neither bulbocavernous nor anal reflexes were detected. Deep tendon reflexes couldn't be detected. Voluntary anal contraction was obtained. The neurological deficit of patient classified by using American Spinal Injury Association impairment Scale (AIS) as T11 AIS-C. Electroneuromyographic (EMG) findings were unremarkable in both nerve conduction studies and needle EMG. Urodynamic study findings were compatible with flaccid neurogenic bladder. According to MRI examination, there was no spinal cord or ligamentous injury. The only findings were of the protrusion of T8-9, T9-10 intervertebral disc material, and obliteration of subarachnoid space (Figure 1, 2). Rehabilitation program performed, including musclestrengthening, balance-training, and gait-training for one hour each day. He learned transfers, then started practicing standing up in the parallel bars with the assistance of the posterior shell orthosis. Clean intermittent catheterization was performed for neurogenic bladder management 6 times per day. According to the follow-up test after four weeks, bulbocavernous reflex returned, and bilaterally achilles clonus were obtained. Manual muscle testing revealed at the time of discharge; hip flexors were of 3/5, knee extensors, were of 4/5, ankle dorsiflexors, plantar flexors, toe extensors were of 2/5, and 1/5, respectively. He was walking with the assistance of the left knee-ankle-foot orthosis (KAFO) and walker.

#### Discussion

SCIWORA was described more prevalent in children due to the increased spinal flexibility and higher head/cervical spinal length ratio, and the difficulties in radiographic assessment of spine in the immature skeleton [1]. Thoracic spinal injury in adults without any vertebral fractures or discoligamentous in-



Figure 1. Midsagittal, T2-weighted MR Scan (TR/TE, 3500 /120) shows that the signal intensity of the thoracic spinal cord is normal. A double, contiguous minimal disc protrusion in the thoracic spine (T8-T9, T9-T10) is seen.



Figure 2. Axial T2-weighted image shows that there is a small left paracentral disc protrusion effacing the subarachnoid space at T8-T9, but the signal intensity of thoracic cord at this level is normal.

jury has been reported very rarely and all MRI investigations showed myelum contusion or cord edema in these reports [3]. SCIWORA in adults without significant spinal cord or ligamentous injury is a very rare described entity in the literature. In our case we reported only intervertebral disc protrusion on MRI examination. Our patient was 27 years old, the spinal canal diameter was normal. MRI findings didn't prove any facilitating factors for SCIWORA as, ligamentum flavum hypertrophy, spinal stenosis or spondylosis. He defined falling from a height of 8 meters and concurrent blunt trauma to the thoracic region. Boese et al. collected the data of SCIWORA in adults consisted of 164 cases by classification with MRI as type I if there were

no detectable abnormalities, type IIa with extraneural abnor-

malities, type II b with intraneural abnormalities, type IIc with

both intraneural and extraneural abnormalities [4]. The authors defined that extraneural abnormalities were protrusion of intervertebral disc or herniation, ligamentum flavum bulging, posterior longitudinal ligament ossification, spondylosis, ligamentous abnormalities, prevertebral soft tissue swelling. They reported 7.1% SCIWORA with no MRI abnormalities. 11.7% with extraneural abnormalities, 36.9% with intraneural abnormalities, and 44.3% with both intraneural and extraneural abnormalities[4]. They reported that AIS D was the neurological severity level which was associated with most favorable outcome [4]. They also examined the relationship between MRI findings and functional outcome. Individuals classified in group type I had significantly better prognosis when compared with type II. In patients with abnormal MRI findings, type IIa was found to achieve more favorable outcomes among type IIa, IIb, IIc [4]. On the other hand, Lui et al. recently reported that the prognosis of SCIWO-RA is not related to MRI findings [5]. Sharma et al. reported that cord edema was related to the best functional outcome while hemorrhage was associated with the worst outcome in adults with SCIWORA [6]. Our patient was AIS C, and type IIa MRI classification, and he achieved partial neurological improvement during rehabilitation.

SCIWORA without any abnormality based on MRI findings is very rarely described in the literature. Recently Park et al. reported a patient with no abnormalities in terms of pathological reflexes, urodynamic findings, and on MRI examination [7]. Delialioglu et al. reported a case SCIWORET with cervicothoracic injury at the level of C7-T1, classified AIS B initially, and progressed to AIS D after the rehabilitation program. In contrast to our report, the MRI demonstrated the myelomalacic field in this report [8].

### Conclusion

The term SCIWORA was defined as SCI by MRI with the absence of fracture or subluxation by plain x-ray and/or CT scan. The relevant group of patients remains a clinical challenge for both neurosurgeons and physiatrists because of the difficulty during the diagnostic process. Moreover the spinal shock period may constitute a suspicion of peripheral nerve lesion or neural root avulsion. Although we had a suspicion of cauda equina syndrome in our case, the absence of peripheral nerve involvement on electrodiagnostic study, the termination of spinal shock, and emerging the signs of upper nerve involvement directed the diagnose to SCIWORA. Diagnose of SCIWORA in adults may take more time than expected. Even the SCIWORA in children is a clinical challenge during the acute process, management of SCIWORA in adults may be more difficult for clinician with the absence of abnormality in MRI.

### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Pang D, Wilberger JE Jr. Spinal cord injury without radiographic abnormalities in children. J Neurosurg 1982;57(1):114-29.
- 2. Saruhashi Y, Hukuda S, Katsuura A, Asajima S, Omura K. Clinical outcomes of cervical spinal cord injuries without radiographic evidence of trauma. Spinal Cord 1998;36(8):567-73.
- 3. Van Buul G, Oner FC. Thoracic spinal cord injury without radiographic abnormality in an adult patient. Spine J 2009;9(3):e5-8.
- 4. Boese CK, Lechler P. Spinal cord injury without radiologic abnormalities in adults: a systematic review. J Trauma Acute Care Surg 2013;75(2):320-30.

- 5. Liu Q, Liu Q, Zhao J, Yu H, Ma X, Wang L. Early MRI finding in adult spinal cord injury without radiologic abnormalities does not correlate with the neurological outcome: a retrospective study. Spinal Cord 2015; DOI: 10.1038/sc.2015.45.
- 6. Sharma S, Singh M, Wani IH, Sharma S, Sharma N, Singh D. Adult Spinal Cord Injury without Radiographic Abnormalities (SCIWORA): Clinical and Radiological Correllations. J Clin Med Res 2009;1(3):165-72.
- 7. Park JW, Lee YG, Choi YH, Seo JW, Lee SM, Kim JI, Ko YJ. Traumatic Atypical Tetraplegia Without Radiologic Abnormalities Including Magnetic Resonance Imaging in an Adult: A Case Report. Ann Rehabil Med 2015;39(1):146-9.
- 8. Delialioglu US, Culha C, Yigit Z, Ozel S, Delialioglu O. Cervicothoracic Spinal Cord Injury Without Radiographic Evidence of Trauma (SCIWORET) in an Adult: A Case Report. Turk J Phys Med Rehab 2007;53(4):164-7.

#### How to cite this article:

Yalbuzdag SA, Kaya T, Sengul İ, Karatepe AG, Adibelli ZH. SCIWORA at Thoracic Level in an Adult: A Case Report. J Clin Anal Med 2015;6(suppl 5): 629-31.



# Acute Respiratory Distress Syndrome and Pulmonary Hypertension Induced By Adenovirus Infection: A Child Case

# Adeno Virüs Enfeksiyonuna Bağlı Gelişen ARDS ve Pulmoner Hipertansiyon : Bir Çocuk Olgusu

Adeno Virüs Enfeksiyonuna Bağlı Gelişen ARDS / ARDS Induced By Adenovirus Infection

Vildan Güngörer

Çocuk Yoğun Bakım Servisi, Samsun Kadındoğun ve Çocuk Hastalıkları Hastanesi, Samsun, Türkiye

#### Özet

Adenovirus, çocuklarda akut solunumsal hastalıklarda major patojenlerden biri olup çift sarmallı DNA virüsüdür Adeno virüs asemptomatikten ölümle sonuçlanan ciddi enfeksiyona kadar çok değişik klinik şekillerde karşımıza çıkmaktadır. Bizim olgumuzda prematüre doğmuş olup uzun süre yenidoğan yoğunbakımda yatmış olan 6ay 18 günlük bilinen immünyetmezliği olmayan bir hastada adenovirüse bağlı ARDS ve pulmoner hipertansiyon sunuldu.

### Anahtar Kelimeler

Adenovirüs; Akut Solunum Yetmezliği; Pnömoni

#### Abstract

Adenovirus is a double-stranded DNA virus and one of the major pathogens in acute respiratory diseases in children. Adenovirus may present in many different clinical forms from asymptomatic to fatal serious infections. The present study reported a case of adenovirus-associated ARDS and pulmonary hypertension in a 6-month and 18-day old non-immunodeficient patient who was born premature and hospitalized for a long time in the neonatal intensive care.

#### Keywords

Adenovirus; Acute Respiratory Distress Syndrome; Pneumonia

DOI: 10.4328/JCAM.3605 Received: 15.05.2015 Accepted: 13.06.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 632-4 Corresponding Author: Vildan Güngörer, Çocuk Yoğun Bakım Servisi, Samsun Kadındoğum ve Çocuk Hastalıkları Hastanesi, 55100, Samsun, Türkiye. GSM: +905069468791 E-Mail: vildan\_61183@hotmail.com

Adenovirus is a double-stranded DNA virus and one of the major pathogens in acute respiratory diseases in children [1]. Adenovirus may present in many different clinical forms from asymptomatic to fatal serious infections. Although rhinitis, conjunctivitis, pharyngitis, tracheitis, gastroenteritis or keratoconjunctivitis are clinically more common, it also leads to lower respiratory tract infections such as bronchiolitis, croup and pneumonia [1-5]. Adenovirus can be very serious and fatal for very young, very old and immunocompromised patients. Adenovirus-associated pneumonia can lead to a spectrum of problems ranging from a typical bronchopneumonia to fatal acute respiratory distress syndrome (ARDS) [2,6-10]. Serological tests, DNA detection by polymerase chain reaction (PCR) and detection of adenovirus-specific antigen are used for diagnostic purposes; however, adenovirus-specific therapy is limited [11-13].

#### **Case Report**

A 6-month and 18-day old baby girl born at 26 weeks weighing 840 grams who was intubated for 78 days and hospitalized for a total of 3 months in the neonatal intensive care unit with the diagnoses of prematurity, respiratory distress syndrome (RDS), retinopathy of prematurity (ROP), bronchopulmonary dysplasia (BPD) was admitted with respiratory distress and cyanosis. The patient's vitals were as follows: body temperature: 38.9°C, blood pressure: 65/30 mmHg, peak heart rate: 165/min, respiration rate: 62/min and So2: 65%. Physical examination indicated that she had tachypnea, subcostal and intercostal retractions, dyspnea and peripheral cyanosis. When listened, lung sounds were coming from deep. Her body weight was 4600 grams (<3p). The results of laboratory analysis were as follows: Hb: 10.7 g/dl, WBC:23790/mm3, PLT:240000/ mm3, and neutrophils: 17030/ mm3. The results of biochemical analysis were as follows: AST: 238 and ALT: 141. The level of C-reactive protein (CRP) was normal. The blood gas values were as follows: pH: 7.28, pCO2: 36 and HCO3: 18. A posteroanterior chest X-ray showed paracardiac infiltrations.

The patient was admitted to the neonatal intensive care with a diagnosis of pneumonia. The monitored patient received normal saline infusion twice due to the low blood pressure. She was administered oxygen therapy using a high-flow oxygen delivery device (flow 15L/min, FiO2: 60%). A broad-spectrum antibiotherapy was initiated (vancomycin + meropenem). Tamiflu was prescribed due to the outbreak of H1N1. Echocardiography showed a secundum atrial septal defect (ASD) of 6.5 mm diameter, tricuspid regurgitation (TY) of 3.5m /sec velocity and pulmonary arterial (PA) pressure of 50mmHg. She was diagnosed with pulmonary hypertension and prescribed enalapril and lasix by pediatric cardiology. The patient's hypoxemia increased despite the high flow of oxygen. The patients with low saturations and respiratory acidosis (pH: 7.21, pCO2: 68) was intubated and put on a ventilator on P-SIMV (Pressure-Synchronized Intermittent Mandatory Ventilation) mode at the eighth hour of hospitalization (PIP: 25, PEEP: 5, rate: 30). Repeated posteroanterior chest X-ray showed a frosted glass pattern associated with ARDS (Figure 1). When the patient's hypoxemia did not improve, PEEP set on the ventilator was gradually increased

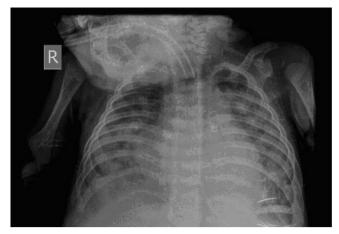


Figure 1. X-ray showed a frosted glass pattern

(PEEP: 7). The patient with a low blood pressure was supported with inotropic agents (adrenaline infusion 0.2mcg/kg/min). An abdominal ultrasonography was conducted on the patient with elevated results of liver function tests. The ultrasonography revealed minimal intraperitoneal fluid. A sputum sample was taken for the Respiratory Virus PCR Panel and a blood sample was also taken for CMV PCR. ARDS began to disappear on the third day of the follow-up. The ventilator settings were gradually reduced and inotropic support was stopped. The patient was extubated on the seventh day of hospitalization. After extubation, the results of respiratory virus panel (RVP) test were PCR positive for adenovirus and PCR negative for cytomegalovirus (CMV). Echocardiography was repeated for control purposes. Pulmonary hypertension disappeared. Minimal pericardial effusion was detected. It was suggested to stop enalapril and lasix. When the liver function tests and chest x-ray of the patent with good oral intake returned to normal, she was discharged on the thirteenth day of hospitalization.

#### Discussion

Adenovirus infections are quite common in children aged between 6 months and 5 years [1]. The most important cause of hospitalization, morbidity and mortality is viral pneumonia especially in premature babies and in the first 6 months of life. Adenovirus is the underlying pathogen in 10% of respiratory diseases in children. Adenovirus infections often present as rhinitis pharyngitis, tracheitis, gastroenteritis or keratoconjunctivitis while the asymptomatic infection rate approaches nearly 50% [2]. Although it is rare, adenovirus leads to severe clinical manifestations such as ARDS, pulmonary hypertension or disseminated infection especially in newborns, premature infants and immunocompromised persons.

The present study reported a case of adenovirus-associated ARDS and pulmonary hypertension in a 6-month and 18-day old non-immunodeficient patient who was born premature and hospitalized for a long time in the neonatal intensive care. In our case, adenovirus was detected by polymerase chain reaction (PCR) method using sputum samples, and other respiratory viruses (human coronavirus 229E, human bocavirus, human coronavirus OC43, parainfluenza 1/2/3, respiratory syncytial virus A/B, rhinovirus, enterovirus, parechovirus, H1N1, influenza A, influenza B, Human metapneumovirus, parainfluenza 4, human coronavirus HKU1, and human coronavirus NL63) were excluded using the same method.

In a study involving 80 adenovirus-infected patients, Chen et al. reported that 17 patients were hospitalized in ICU and only one case who was a 1.7-year-old boy with cerebral palsy had ARDS and resulted in death [14]. In another study, only 9 of 617 adenovirus-infected children had serious infection and only one died [15]. Our patient had a history of premature birth and no underlying disease. In the literature, there are disseminated cases of ARDS and fatal cases of adenovirus infection especially in immunocompromised patients and newborns [16-17]. However, it is a rare case that adenovirus-associated ARDS and pulmonary hypertension developed in a patient who had no known disease but a history of premature birth 6 months.

Previous studies have reported fever, cough and rhinorrhea that are typical symptoms of adenovirus-associated pneumonia as well as tachypnea and dyspnea in nearly half of patients [18-21]. In our case, the most obvious symptom was tachypnea and dyspnea. There was also a high fever and a high level of leukocytes. In the study of Chen et al., one quarter of the patients had high levels of leukocyte in a similar way to bacterial infection while half of the patients had high levels of CRP and a high fever [14]. However, in our case, the level of CRP was not markedly high. Although the chest X-ray revealed no obvious infiltration, our patient was prescribed a broad-spectrum antibiotic as the high level of leukocyte and high fever initially suggested bacterial pneumonia. In several studies in the literature, clinicians have difficulty in distinguishing between bacterial infection and adenoviral infection due to similarities in symptoms [14-19]. We believe that the reason for prescribing antibiotics in many hospitalized patients is the clinical confusion of diagnosis. Our patients had also symptoms of acid in the abdomen and minimal pericardial effusion. Previous studies have not reported such symptoms in adenovirus infections.

To conclude, although adenovirus infections rarely show a serious clinical course in healthy children, it should be remembered that it is an infection that can even result in death, and health care providers should be particularly vigilant in adenovirus-associated pneumonia. Adenovirus-associated pneumonia should be considered especially in patients who do not respond to antibiotics as it clinically mimics bacterial pneumonia.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Cherry JD. Adenoviruses. In: Feigin RD, Cherry JD, Demmler GJ, Kaplan SI, editors. Textbook of pediatric infectious diseases. 6th ed. Philadelphia: WB Saunders; 2009.p.1843-62.
- 2. Horwitz M. Adenoviruses. In: Fields B. Knipe D.Howley P. Chanok R. Melnick I. Monath T, Roizman B, Straus S, editors. Virology. 3rd ed. Philadelphia: Lippincott-Raven Publishers: 1996.p.2149-63.
- 3. Pancini DL, Collier AM, Henderson FW. Adenovirus infections and respiratory illness in children in group day care. J Infect Dis 1987;156(6):920-7.
- 4. Edwards KM, Thompson J, Paolini J, Wright PF. Adenovirus infections in young children. Pediatrics 1985;76(3):420-4.
- 5. Dominguez O, Rojo P, de Las HS, Folgueira D, Contreras JR. Clinical presentation and characteristics of pharyngeal adenovirus infections. Pediatr Infect Dis J 2005;24(8):733-4.
- 6. Louie IK, Kaion AE, Holodniv M, Guardia-LaBar L, Lee B, Petru AM, et al. Severe pneumonia due to adenovirus serotype 14: a new respiratory threat? Clin Infect Dis 2008:46(3):421-5
- 7. Chuang YY, Chiu CH, Wong KS, Huang JG, Huang YC, Chang LY, et al. Severe adenovirus infection in children. J Microbiol Immunol Infect 2003;36(1):37-40.
- 8. Carballal G, Videla C, Misirlian A, Requeijo PV, Aguilar Mdel C. Adenovirus type

- 7 associated with severe and fatal acute lower respiratory infections in Argentine children. BMC Pediatr 2002;2(1):6
- 9. Lang WR, Howden CW, Laws J, Burton JF. Bronchopneumonia with serious sequelae in children with evidence of adenovirus type 21 infection. Br Med J 1969;1(5636):73-9.
- 10. Kuo CY, Lee CY, Kao CL, Hsu YH. A fatal case of viral pneumonia in a child infected with adenovirus type 3. Acta Paed Sin 1990;31(1):40-5.
- 11. Birenbaum E. Linder N. Varsanon A. Azar R. Kuint I. Spierer A. et al. Adenovirus type 8 conjunctivitis outbreak in a neonatal intensive care unit. Arch Dis Child 1993;68(5 Spec No):610-1.
- 12. Uhnoo I, Wadell G, Svensson L, Johansson ME. Importance of enteric adenoviruses 40 and 41 in acute gastroenteritis in infants and young children. J Clin Microbiol 1984;20(3):365-72.
- 13. Van R, Wun CC, O'Ryan ML, Matson DO, Jackson L, Pickering LK. Outbreaks of human enteric adenovirus types 40 and 41 in Houston day care centers. J Pediatr 1992:120(4):516-21
- 14. Chen SP, Huang YC, Chiu CH, Wong KS, Huang YL, Huang CG et al. Clinical features of radiologically confirmed pneumonia due to adenovirus in children, lournal of Clinical Virology 2013:56(1):7-12.
- 15. Chuang YY, Chiu CH, Wong KS, Huang JG, Huang YC, Chang LY, et al. Severe adenovirus infection in children. J Microbiol Immunol Infect 2003;36(1):37-40.
- 16. Sürmeli-Onay Ö, Karhan A, Güçer Ş, Karagöz T, Yurdakök M. Postmortem tanı alan fatal adenoviral pnömoni: Bir yenidoğan olgusu. Çocuk Sağligi ve Hastaliklari Dergisi 2012;55(2).
- 17. Pinto A, Beck R, Jadavji T. Fatal neonatal pneumonia caused by adenovirus type 35: report of one case and review of the literature. Arch Pathol Lab Med 1992;116(1):95-9.
- 18. Hong JY, Lee HJ, Piedra PA, Choi EH, Park KH, Kho YY, et al. Lower respiratory tract infections due to adenovirus in hospitalized Korean children: epidemiology, clinical features, and prognosis. Clin Infect Dis 2001;32(10):1423-9.
- 19. Lin CH, Huang YC, Chiu CH, Huang CG, Tsao KC, Lin TY. A cluster of adenovirus serotype 3 infections in children in northern Taiwan: clinical features and laboratory findings. J Microbiol Immunol Infect 2007;40(4):302-9.
- 20. Assaad F, Borecka I. Nine-year study of WHO virus reports on fatal viral infections. Bull World Health Organ 1977;55(4):445-53.
- 21. Teper AM, Kofman CD, Maffey AF, Vidaurreta SM. Lung function in infants with chronic pulmonary disease after severe adenoviral illness. J Pediar 1999;134(6):730-3.

#### How to cite this article:

Güngörer V. Acute Respiratory Distress Syndrome and Pulmonary Hypertension Induced By Adenovirus Infection: A Child Case. J Clin Anal Med 2015;6(suppl 5):



# **Evaluation of Alone or Combined Colistin Therapy Success in** Patients with Carbapenem-Resistant Acinetobacter Pneumonias

Acinetobacter Pnömonisinde Kombine Kolistin / Combined Colistin in Acinetobacter Pneumonia

Özlem Erçen Diken<sup>1</sup>, Ayhanım Tümtürk<sup>2</sup>, Sertaç Arslan<sup>1</sup> <sup>1</sup>Chest Diseases Department, Hitit University, <sup>2</sup>Infection Diseases, Corum State Hospital, Corum, Turkey

Amaç: Karbepenem dirençli gram negatif bakteri yoğun bakım ünitelerinde artan bir tehlikedir. Kliniğimizde acinetobacter enfeksiyonunda farklı tedavi rejimlerinin ve mortalite oranlarının sonuçlarını saptamayı amaçladık. Kombine kolistin tedavisi üstün mü, değil mi? Gereç ve Yöntem: Ocak 2013 ve Haziran 2014 tarihleri arasında kliniğimizde tanı konulmuş ve tedavi edilmiş, acinetobacter enfeksiyonu olan 23 olgu, tedavileri ve mortalite süreleri yönünden retrospektif olarak incelenmiştir. Bulgular: 23 hastanın 19'u yoğun bakımda, 4'ü serviste tedavi edilmiştir. Yoğun bakım ünitesindeki 19 hastadan; sefoperazon+sulbaktam duyarli olan grupta 2 hasta, sefoperazon+sulbaktama da duvarli olmavan grupta 3 hasta kolistin monoterapisi ile exitus oldular. Kombine tedavi alan hastalar arasında, 2 olguya amikasin+kolistin kombine tedavisi verildi; bu iki hasta da exitus oldular. Kolistin+rifampisin kombine tedavisi alan bir olgu da iyi yanıt alındı. Serviste tedavi edilen 4 hastanın 3'ü karbapenem dirençliydi ve bunlardan da 1'i sadece kolistine duyarlıydı. Bu hasta, kolistin+tigesiklin kombine tedavisi aldı ve iyi yanıt elde edildi. Tartışma: Akciğerin acinetobacter enfeksiyonlarında mortaliteyi azaltmak için kombine kolistin tedavisi yararlı görünmektedir. Karbapenem dirençli acinetobacter olgularının rifampisin ve tigesiklin kombinasyonu açısından değerlendirilmesi gerektiği görüşündeyiz.

#### Anahtar Kelimeler

Acinetobacter; Rifampisin; Tigesiklin

Aim: Carbapenem-resistant gram negative bacteria represent an increasing problem worldwide for intensive care units. We aimed to detect the outcome of different treatment regimens and mortality rates of Acinetobacter infection in our clinic. Is combined colistin therapy superior or not? Material and Method: 23 cases diagnosed and treated in our unit with documented Acinetobacter infections between January 2013 and June 2014 were retrospectively evaluated in terms of treatments administered and mortality rates. Results: 19 of 23 patients were treated in ICU and 4 of 23 patients were treated in normal patient ward. Among 19 ICU patients, 2 patients in cefoperazone + sulbactam - susceptible groups and 3 patients with cefaperazone + sulbactam - resistant groups died with colistin monotherapy. Among patients receiving combined treatment, two patients were given amikacin + colistin combination and they both died. A single patient receiving colistin + rifampicin combination responded well. Of the 4 patients treated in the normal patient ward, 3 had carbapenem resistance, and of these, only 1 was susceptible to colistin. This patient received colistin + tigecycline combination and had good response. Discussion: The combined colistin treatment seems plausible in terms of reducing mortality in Acinetobacter infections of lung. We believe that in patients with carbapenem-resistant Acinetobacter infections, a consideration should be given to rifampicin or tigecycline combination with colistin.

Acinetobacter; Colistin; Rifampicin; Tigecyclin

DOI: 10.4328/JCAM.3654 Corresponding Author: Özlem Erçen Diken, Chest Diseases Department, Hitit University, 19000, Çorum, Turkey. T.: +90 3642230300 F.: +90 3642230323 E-Mail: oercen@hotmail.com

Infections due to carbapenem-resistant gram negative bacteria (CRGNB) represent an important problem worldwide, and the main question regarding the treatment of this patient group is whether a combination treatment or monotherapy should be administered. Despite some reports suggesting a superiority of combination regimens over colistin monoteraphy in CRGNB infections, therapeutic guidelines provide no definitive approach in such cases [1]. After the initial observations suggesting better outcomes in patients with pneumonia due to carbapenemresistant Acinetobacter who were treated with combined regimens, we decided to undertake a study comparing different regimens in our clinic and assessing effect of combined treatment and monotherapy on morbidity and mortality.

### Material and Method

Patients diagnosed and treated in our unit with documented Acinetobacter infections between January 2013 and June 2014 were retrospectively evaluated in terms of treatments administered and mortality rates. Of the 23 cases overall, 19 were treated in the intensive care unit, whereas 4 were admitted to normal patient ward. Microbiological cultures were obtained and antibiograms were performed in tracheal aspiration or sputum samples. Mortality, time to mortality, growth in microbiological samples, time to documented absence of growth in sputum samples, c-reactive protein (CRP) measurements, fever, and duration of follow-up after discharge were recorded. Due to small sample size formal statistical tests were not performed.

#### Results

All cases, i.e. 19 patients, with Acinetobacter infections in the intensive care unit subsequently proved to have ventilator - associated pneumonia. Among the isolated species from these 19 patients, only 2 (11%) were susceptible to carbapenem. Therefore, these two patients responded to a course of carbapenem and have now completed 1 year of follow - up.

Of the 17 cases (89%) with carbapenem - resistant Acinetobacter infection, 11 (64%) were susceptible to cefaperazone + sulbactam. Among these 11 cases with cefaperazone + sulbac-

Table 1. The treatments and mortality in patients with carbapenem-resistant Acinetobacter infection in intensive care unit

Case	Threat- ment	Sputum culture	CRP response	Complica- tions	Mortality	Mortality day*	Follow- up
1.	colistin + rifampicin	8.day, no m.o.	yes	no	no	-	1-year of follow-up
2.	colistin	-	-	-	yes	1.day	
3.	colistin + amikacin	3.day, no acineto- bacter, but MRSA	no	renal failure	yes	5.day	
4.	colistin + amikacin	9.day, no m.o.	no	no	yes	9.day	
5.	colistin	4.day, no m.o.	no	renal failure / electrolyte disturbance	yes	4.day	
6.	colistin	-	-	-	yes	1.day	

<sup>\*:</sup> after threatment

MRSA: Methicillin-resistant Staphylococcus aureus

tam - susceptible but carbapenem- resistant cases, 2 died following colistin monotherapy, while 2 died before a treatment was given. Of the latter two cases, the results of the sputum cultures could only be available on the day of death precluding any treatments. Of these 11 patients with cefaperazone + sulbactam-susceptible but carbapenem - resistant infection, one died despite treatment with cefaperazone + sulbactam. Following CRP and clinical response, this case had resurgence of CRP. and probably died after a new infection before the causative agent was detected. In 6 of the 11 cases with cefoperazone + sulbactam - susceptible but carbapenem - resistant infection, a response to cefoperazone + sulbactam could be obtained with subsequent discharge.

The remaining 6 cases (35%) with carbapenem - resistant Acinetobacter had cefoperazone + sulbactam resistant infection. Of these, 4 were susceptible to colistin only (percentage of cases with carbapenem - resistance and exclusive colistin - susceptibility, 24%) while 2 also had amikacin susceptibility. Of the 6 cases with carbapenem - resistant and cefoperazone + sulbactam resistant Acinetobacter infection, one received colistin + rifampicin combination, who had no growth in the sputum culture on day 8, with reduction in CRP and subsequent discharge. The patient has now completed 1 - year of follow-up. The second case died on the first day of colistin monotherapy. The third case received colistin + amikacin due to susceptibility to both amikacin and colistin. Although there was no growth on day 3 of the treatment, CRP did not decrease and no clinical response to treatment was observed. A repeat sputum culture revealed meticilline - resistant Staphylococcus aureus (MRSA) and the patient died on the day of documented MRSA. This patient also had signs of renal failure. The fourth case had amikacin susceptible Acinetobacter infection and received colistin + amikacin combination. On Day 9 of the treatment there was no growth in the sputum culture, despite no decrease in CRP. This patient also died. The fifth case received colistin monotherapy. with no CRP response and no growth in sputum culture on Day 4. He developed renal toxicity and electrolyte disturbance, attributed to antibiotic treatment, and died following cardiac arrest. Again, microbiological growth was detected in the sixth

> case on the day the patient died due to cardiac arrest developing at Day 1 of treatment. Table 1 shows the treatments and mortality in patients with carbapenem - resistant Acinetobacter infection.

> Of the 4 patients admitted to the normal patient ward, 3 had carbapenem resistant Acinetobacter. One of these cases had meropenem susceptible infection and he was receiving concomitant antituberculous treatment. He is currently being followed - up by an outpatient tuberculosis clinic. Two patients had cefoperazone + sulbactam susceptible infections and were discharged following successful treatment with cefoperazone + sulbactam. Among the three carbapenem-resistant cases in the normal patient ward group, one only had colistin susceptibility and received colistin and tigecycline. The patient was also receiving concurrent hemodialysis. At day 10 there was no growth in

<sup>:</sup> m.o: microorganisms

CRP: C-Reactive Protein

the sputum culture and he was eventually discharged. All patients who received combined colistin therapy or colistin monotherapy are listed in Table 2.

Table 2. The treatments and mortality in patients who receive combined colistin therapy or colistin monotherapy

		intensive (				
Threatment		cefaperazone + sulbactam- susceptible	cefoperazone + sulbactam resistant	normal patient ward**	Mortality	Total
mono therapy	colistin	2 case	3 cases*	-	yes	5 cases
combined therapy	colistin/ amikacin	-	2 cases	-	yes	4 cases
	Colistin/ rifampicin	-	1 case	-	no	
	colistin/ tigecycline	-	-	1 case	no	

<sup>\*:</sup> Two cases died on the first day of colistin monotherapy

#### Discussion

In a recent review examining the treatment of Acinetobacter infections, three studies have been found that demonstrated a superiority of combination treatment over monotherapy in multiple-drug resistant Acinetobacter including carbapenem. The authors have concluded that combined treatment may be recommended for severe disease [2]. In our patient group, 5 subjects had monotherapy as compared to 4 patients receiving combined treatment. Of the combinations, 2 involved amikacin, and one of these patients died with electrolyte disturbances and renal toxicity. One case had colistin plus rifampicin and 1 had colistin plus tigecycline combination, with good response. On the other hand, all of the five patients receiving monotherapy died.

Again in another recent publication, the lack of data demonstrating the efficacy of combination therapy has been emphasized, despite the common use of combinations in the clinical practice [3]. In that paper, data obtained across 28 Spanish hospitals were analyzed. Most of the patients involved in the assessment were receiving mechanical ventilation (68.6%) and the most common type of infection was pneumonia (50.5%). The agents that were most commonly used in combinations were colistin (67.6%) and carbapenem, while the combination utilized with the highest frequency was colistin-tigecycline (27.3%). The observed 30 - day mortality rates in monotherapy and combination therapy were 23.5% an 24.2% (p = 0.94), showing no mortality benefit with combination. On the other hand, among our patients with carbapenem resistant Acinetobacter who received colistin (n = 9), one (11.1%) had colistin plus tigecycline and one (11.1%) had colistin plus rifampicin, and both cases responded well to these regimens. The mortality rate in 5 patients (55%) receiving monotherapy was 100%. Although it is not possible to draw firm conclusions due to small sample size, our observations suggest a better efficacy for the combination.

#### Conclusion

In multiple drug resistant Acinetobacter pneumonia the mortal-

ity rate is high and colistin currently represents the last resort. A consideration should be given to rifampicin plus tigecycline combination in carbapenem resistant Acinetobacter infections.

> Further studies are warranted to better define the role of combinations and to establish standard protocols.

> Authors' contributions: ÖED planned the study, collected data and wrote the paper. AT and SA helped to collect data and write the paper.

### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Paul M, Carmeli Y, Durante-Mangoni E, Mouton JW, Tacconelli E, Theuretzbacher U et al. Combination therapy for carbapenem-resistant Gram-negative bacteria. J Antimicrob Chemother 2014;69:2305-
- 2. Poulikakos P, Tansarli GS, Falagas ME. Combination antibiotic treatment versus monotherapy for multidrug-resistant, extensively drug-resistant, and pandrug-resistant Acinetobacter infections: a systematic review. Eur I Clin Microbiol Infect Dis 2014: 33(10):1675-

85.

3. López-Cortés LE, Cisneros IM, Fernández-Cuenca F, Bou G, Tomás M, Garnacho-Montero J et al. Monotherapy versus combination therapy for sepsis due to multidrug-resistant Acinetobacter baumannii: analysis of a multicentre prospective cohort. J Antimicrob Chemother 2014;69:3119-26.

#### How to cite this article:

Diken ÖE, Tümtürk A, Arslan S. Evaluation of Alone or Combined Colistin Therapy Success in Patients with Carbapenem-Resistant Acinetobacter Pneumonias. J Clin Anal Med 2015;6(suppl 5): 635-7.

<sup>\*\*:</sup> One patients had karbapenem and cefoperazone + sulbactam resistant infections. The other 3 patients in normal patient ward had karbapenem or cefoperazone + sulbactam susceptible and were discharged following successful treatment with karbapenem or cefoperazone + sulbactam.



# Agammaglobulinemia in a Patient with Smith-Lemli-Opitz Syndrome: Case Report

# Agamaglobulinemi ile Seyreder Smith-Lemli-Opitz Sendromu: Olgu Sunumu

Smith-Lemli-Opitz Sendromu ve Agamaglobulinemi / Smith-Lemli-Opitz Syndrome and Agammaglobulinemia

H. Haluk Akar¹, Songül Gökay², Turkan Patiroglu¹, Deniz Okdemir³, Mustafa Kendirci²
¹Department of Pediatric Immunology, ²Department of Pediatric Metabolism,
³Department of Pediatric Endocrinology, Erciyes University School of Medicine, Kayseri, Turkey

#### Ozet

Smith-Lemli-Opitz sendromu (SLO) otozomal resesif kalıtımla geçen multipl kaonjenital anomaliler, mikrosefali, müsküler hipotoni ve ağır gelişimsel gerilikle seyreden kalıtsal bir hastalıktır. Bu sendroma 7-dehidrokolesterol redüktaz enzimindeki eksiklik neden olur. SLO hastaları müsküler hipotonisitenin neden olduğu azalmış motilite ve solunum eforundan kaynaklanan tekrarlayan solunum yolu enfeksiyonları gösterirler. Bu çalışmada tekrarlayan üriner sistem enfeksiyonları gösteren, kronik diyaresi olan ve rektal sürüntü kültürlerinde Klepsiella pnomonia pozitifliği saptanan 1 yaşında bir erkek hastada sunulmuştur. Hasta aynı zaman da immunglobulin G (IgG) değerleri 50-100 mg/dL arasında olacak şekilde belirgin agamaglobulinemiye sahipti. Hastanın takiplerinde intavenöz immünoglobulin replasmanı ile belirgin klinik iyileşme gözlendi. Bu çalışma ile SLO hastalarında tekrarlayan enfeksiyonların görülmesi ile altta yatan bir immün yetmezliğin olabileceği akılda tutulması gerektiği vurgulanmak istenmiştir.

### Anahtar Kelimeler

X Geçişli Agamaglobulinemi; Smith-Lemli-Opitz Sendromu; Rekürrent Enfeksiyonlar

#### Abstract

Smith-Lemli-Opitz syndrome (SLO) is a rare autosomal recessive (AR) inherited genetic disorder characterized by multiple congenital anomalies, microcephaly, muscular hypotonia, and severe developmental delay. The deficiency of 7-dehydrocholesterol reductase enzyme leads to this syndrome. Patients with SLO display recurrent respiratory infections due to secondary muscular hypotonia which leads to decreased motility and respiratory effort. In this study, we report a 1-year-old boy with SLO presented with recurrent urinary infections and chronic diarrhea with Klebsiella pneumonia positivity in the rectal swabs. The patient had also markedly decreased immunoglobulin G (IgG) between 50-100 mg/dL. In follow-up of patient, markedly clinical improvement was observed with intravenous immunoglobulin (IVIG) replacement. With this study, we would like to draw attention; recurrent infections may indicate primary immunodeficiencies such as agammaglobulinemia in patients with SLO.

#### Keywords

X Linked Agammaglobulinemia; Smith-Lemli-Opitz Syndrome; Recurrent Infections

DOI: 10.4328/JCAM.3687 Received: 23.06.2015 Accepted: 12.07.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 638-40 Corresponding Author: Haluk Akar, Pediatric Immunology, Erciyes University Medical Faculty, Kayseri, Turkey.
T.: +90 3522076666/25300 F.: +90 3524375825 E-Mail: himmetakar@gmail.com

SLO is an AR inherited genetic disorder caused by 7-dehydrocholesterol (7-DHC) reductase enzyme deficiency in the cholesterol biosynthesis. The incidence of SLO syndrome is estimated as 1/20000-1/70000 [1]. Patients with SLO are characterized by characteristic face appearance with microcephaly, ptosis, anteverted nares and micrognathia, severe developmental delay, and skeletal abnormities such as syndactyly, postaxial polydactyly. Patients with SLO have markedly decreased cholesterol and markedly elevated 7-DHC levels. 7-DHC levels are also used as a biomarker in diagnosis of patients with SLO [1-2]. The DHCR7 gene is located on chromosome 11q12-13 identified as causative gene [3]. These patients present with recurrent respiratory infections due to secondary to muscular hypotonia which leads to decreased respiratory effort. Primary immunodeficiencies have been rarely reported in the SLO patients [4]. Herein, we reported a 1-year-old boy with SLO presented with recurrent urinary infections and chronic diarrhea with K. pneumonia positivity in the rectal swaps. The patient had also agammaglobulinemia.

### Case Report

A 1-year-old boy was born as a first child of unconsanguineous parents. The pregnancy was normal in term of fetal movements. The patient was born as a preterm and small for gestational age (SGA) baby with weight: 1730 gram (< 3p), length: 39 cm (< 3p), and head circumference: 29 cm (<3p) at the 35 weeks of gestation age. At the 3 months of age, he was referred to our clinic for diagnostic purposes. Physical examination revealed hypotonia, bitemporal narrowing, ptosis, prominent eyes, short nose, bilateral low-seated ears, very high upper palate, microretrognathia, anteverted nares, bilateral partial syndactylia between the 2nd and 3rd toes, and scrotal hypoplasia (Fig.1). In his laboratory investigation, serum cholesterol and 7-DHC levels were found as 10 (140-200 mg/dl) and 6.6 (normal range, 0.10 ± 0.05 mg/dl) respectively. Clinical manifestations and markedly decreased cholesterol levels as well as markedly increased 7-DHC levels indicated SLO in the presented patient. Cranial MRI (magnetic resonance imaging) showed partial corpus callosum agenesia. At the 4 months of age, the percutaneous endoscopic gastrostomy was performed due to oral feeding difficulty. In his follow-up, recurrent urinary infections were seen and he was hospitalized 3 times by one year. Abdominal ultrasound and voiding cysto-urethrography were normal. Also he had chronic diarrhea, several times K. pneumonia was cultured in the rectal swabs. At one year of age, immunologic evaluation was found as follows; absolute neutrophil count (ANC): 2060/ mm<sup>3</sup> (1500-400), absolute lymphocyte count (ALC): 4500/mm<sup>3</sup> (2200-8100), CD3: 3900/mm<sup>3</sup> (1300-6000), CD4: 2600/mm<sup>3</sup> (700-4500), CD8: 1270/mm<sup>3</sup> (400-3200), CD19: 100/mm<sup>3</sup> (500-3600), NK: 530/mm<sup>3</sup> (200-1300), IgG: 56 mg/dL (642-788), IgA: 6.2 mg/dL (43-53), IgM: 12 mg/dL (79-110), IgE: 4.2iu/mL (0-50). Genetic test revealed compound heterozygous mutations (c.278 C>T and IVS7+1 G>A) in exon 9 in the 7-DHC reductase (DHCR7) gene (Table 1). However, we couldn't confirm agammaglobulinemia with genetic test. Immunologic and clinical manifestations indicated x-linked agammaglobulinemia in the presented patient. Also, the older brother of mother had recur-

Table 1. Immunologic findings of patient

	Patient	Normal range
ANC (mm³)	2060	1500-400
ALC(mm³)	4500	2200-8100
CD3 (mm³)	3900	1300-6000
CD4 (mm³)	2600	700-4500
CD8 (mm³)	1270	400-3200
CD19 (mm³)	100	500-3600
NK (mm³)	530	200-1300
IgG (mg/dL)	56	642-788
IgA (mg/dL)	6.2	43-53
IgM (mg/dL)	9.2	79-110
IgE (iu/ml)	12	0-50

ANC: Absolute neutrophil count, ALC: Absolute lymphocyte count, NK: Natural killer cell





Figure 1. At the age of 1 year. The appearance of patient was consistent with SLO characteristic microcephaly, bitemporal narrowing, microretrognathia, prominent eyes, ptosis, anteverted nares, bilateral low-seated ears, and bilateral partial syndactylia between the 2nd and 3rd toes

rent pneumonia and he had been interned 3 times which was consistent with agammaglobulinemia.

Oral cholesterol supplementation was initiated as 50 mg/kg/ day and gradually increased to 100 mg/kg/day in one month. Also, intravenous immunoglobulin (IVIG) replacement was started as monthly infusion. After the IVIG replacement therapy K. pneumonia positivity and recurrent urinary infections were not observed in follow-up of the patient.

#### **Discussion**

Cholesterol is a basic cellular membrane lipid molecule. It is highly important membrane permeability and intracellular signaling. Cholesterol is also involved in the synthesis of bile acids, steroid hormones, and vitamin D. In patient with SLO, it is thought that pathogenesis of multisystem manifestations may due to cholesterol deficiency, toxic effects of 7-DHC and its compounds or a combination all of these factors. The diagnosis of SLO based on clinical manifestations and serum cholesterol and 7-DHC levels. Although patients with SLO usually have markedly decreased cholesterol, sometimes mild phenotypic SLO patients may have normal cholesterol levels [1-2]. Some genetic syndromes such as x-linked chondrodysplasia punctata, CHILD syndrome, SC4MOL deficiency, Antley-Bixler syndrome, and HEM dysplasia are involved in cholesterol deficiency which characterized by multisystem malformations. These disorders have quite different manifestations and unique accumulation of biochemical metabolites which are important the pathogenesis

of these disorders. In contrast to these disorders, patients with SLO have markedly elevated 7-DHC levels. Elevated 7-DHC levels are more specific to the patients with SLO than the others [2, 5]. Patients with SLO have an increased number of infections including otitis media, skin and lower respiratory infections due to secondary muscular hypotonia in childhood period. Although patients with SLO often have gastro-esophageal reflux and hypotonia, aspiration pneumonia is rarely seen in their follow-up due to excessive gag reflex [6].

In the medical literature, two articles were reported associated with primary immunodeficiencies in patients with SLO. In the first article, defective monocyte oxidative metabolism was described in a patient presented with recurrent wheezing and atopic dermatitis in 1992 [7]. In the second article, selective antibody immune deficiency against to the polysaccharide antigens was reported in a patient presented with recurrent upper respiratory infection in 2005 [5]. Actually, patients with SLO present with recurrent respiratory tract infections due to secondary to muscular hypotonia which causes decreased respiratory effort. To our knowledge, the presented case is the first agammaglobulinemic patient with SLO.

In conclusion, primary immunodeficiencies have been rarely reported in patients with SLO in the medical literature. In the presented case report, the association of SLO and agammaglobulinemia may be coincidental. However, this case indicates once again that detailed immunologic evaluation is required in patients with SLO who presented with recurrent infections.

### Conflict of interest

The authors declare that they have no conflict of interest

#### References

- 1. Porter FD. Smith-Lemli-pitz syndrome: pathogenesis, diagnosis and management. Eur J Hum Genet 2008;16(5):535-41.
- 2. Demirdöven M, Yazgan H, Korkmaz M, Gebeşçe A, Tonbul A. Smith-lemli-opitz syndrome: a case with annular pancreas. Case Rep Pediatr 2014;2014:623926.
- 3. Wassif CA, Maslen C, Kachilele-Linjewile S, Lin D, Linck LM, Connor WE, et al. Mutations in the human sterol delta7-reductase gene at 11q12-13 cause Smith-Lemli-Opitz syndrome. Am J Hum Genet 1998;63(1):55-62.
- 4. Babovic-Vuksanovic D. Jacobson RM, Lindor NM, Weiler CR, Selective antibody immune deficiency in a patient with Smith-Lemli-Opitz syndrome. J Inherit Metab Dis 2005:28(2):181-6.
- 5. DeBarber AE, Eroglu Y, Merkens LS, Pappu AS, Steiner RD. Smith-Lemli-Opitz syndrome. Expert Rev Mol Med 2011;13:e24.
- 6. Kelley RI, Hennekam RC. The Smith-Lemli-Opitz syndrome. J Med Genet
- 7. Ostergaard GZ, Nielsen H, Friis B. Defective monocyte oxidative metabolism in a child with Smith-Lemli-Opitz syndrome. Eur J Pediatr 1992;151(4):291-4.

#### How to cite this article:

Akar H.H, Gökay S, Patiroglu T, Okdemir D, Kendirci M. Agammaglobulinemia in a Patient with Smith-Lemli-Opitz Syndrome: Case Report. J Clin Anal Med 2015;6(suppl 5): 638-40.



# A Rare Case of Acute Intestinal **Obstruction Due to the Migration of Penile Prosthesis**



Savaş Baba, Osman Yıldırım, Murat Özgür Kılıç, Nermin Damla Hatipoğlu, Cem Emir Güldoğan Clinic of General Surgery, Numune Education and Research Hospital, Ankara, Turkey

An abstract of this manuscript was presented in 15. National Colon and Rectum Surgery Congress as a poster.

Akut barsak tıkanıklığı genel cerrahi pratiğinde en sık acil hastalıklardan biridir. Ameliyat sonrası yapışıklıklar, fıtıklar ve kanserler barsak tıkanıklığı olan olguların çoğunluğunu oluşturmaktadır. Ancak, şişirilebilir penil protez cihazlarına bağlı ince barsak tıkanması son derece nadir bir durumdur. 59 yaşındaki erkek hasta karın ağrısı, bulantı, kusma ve ishal ile acil servise başvurdu. Hastanın klinik ve ultrasonografik bulguları perfore apandisit ile uyumlu idi. Operasyonda, migrate olmuş penil protez rezervuarı ile ileum arasında fibröz bantın neden olduğu ince barsak tıkanması tespit edildi. Hastaya barsak rezeksiyonu olmaksızın bridektomi uygulandı. Şişirilebilir penil protez medikal tedaviye dirençli erektil disfonksiyonu olan hastalar için yaygın olarak kullanılır. Nadir olmakla birlikte, bu tür hastalarda, barsak tıkanıklığının bir nedeni olarak penil protez her zaman akılda tutulmalıdır. Bu nedenle, bu hastalar preoperatif ayrıntılı değerlendirilmelidir.

#### Anahtar Kelimeler

Barsak Tıkanıklığı; İleus; Komplikasyon; Penil Protez

#### Abstract

Acute intestinal obstruction is one of the most common emergent disorders in general surgery practice. Postoperative adhesions, hernias and cancers constitute the majority of cases with intestinal obstruction. However, small bowel obstruction due to an inflatable penile prosthetic device is an extremely rare condition. A 59-year-old male patient was admitted to emergency room with abdominal pain, nausea, vomiting and diarrhea. Clinical and ultrasonograpic findings were consistent with perforated appendicitis. At operation, a small bowel obstruction caused by a fibrous strand between migrated reservoir of the penile prosthesis and ileum was detected. Bridectomy without resection of bowel loop was performed for the patient. Inflatable penile prosthesis is widely used for the patient with erectile dysfunction refractory to medical treatment. Although rare, a penile prosthesis should always be kept in mind as a cause of intestinal obstruction in such cases. Therefore, these patients should be evaluated in detail preoperatively.

#### Keywords

Complication; Ileus; Intestinal Obstruction; Penile Prosthesis

Corresponding Author: Murat Özgür Kılıç, Clinic of General Surgery, Numune Education and Research Hospital, Ankara, Turkey. GSM: +905053573122 E-Mail: murat05ozgur@hotmail.com

Acute intestinal obstruction (AIO) is one of the most common emergent conditions in general surgery practice worldwide, accounting for approximately 15% of all acute abdomen cases [1]. Although postoperative adhesions, obstructed hernias and cancers are the most common causes of AIO, a variety of rare conditions can result in this clinical entity. Among those, migration of penile prosthesis into the abdomen is extremely rare. As is well known, inflatable penile prosthesis has been widely used for men with erectile dysfunction. Several complications such as infection, reservoir herniation, and injury of the surrounding tissues and organs have been described to date. However, best of our knowledge, only a few cases of AIO due to penile implants were reported in the literature [2-4]. There is also no report on this topic from Turkey. In this paper, a rare case of small bowel obstruction due to the migration of penile prosthesis was presented and discussed with the relevant literature.

#### Case Report

A 59-year-old man presented to the emergency room with abdominal pain, nausea, vomiting and minimal distention for three days. He also suffered from diarrhea for the last two days. The patient had several co-existing clinical disorders including diabetes mellitus and coronary artery disease, which were under medical treatment for a long time. He also had a history of penile prosthesis surgery due to diabetic erectile dysfunction 12 years ago. On physical examination, there were tenderness, defense and rebound tenderness at the lower part of abdomen. Routine laboratory tests showed increased inflammatory markers (WBCs: 14.0 K/ µl, CRP: 94.4 mg/l). Abdominal X-ray demonstrated moderate nonspecific dilations of the small bowel, with no pneumoperitoneum. On ultrasonography, a 8 cm tubular structure without response to compression and an amount of fluid within the small bowels were detected, which strongly suggested a perforated appendicitis. At laparotomy via lower midline incision, it was seen that the reservoir of the penile prosthesis migrated into the abdomen and a rigid fibrous strand was present between the reservoir and terminal ileum, which caused a nearly complete intestinal obstruction (figure 1A,B).



Figure 1. A. Penile prosthesis device migrated into the abdominal cavity B. The view of the ileal part of the firm fibrous band after bridectomy. The dilated small bowel proximal to fibrous strand can be also clearly seen.

The appendix was dilated and hyperemic due to intra-abdominal fluid. There was no severe ischemic damage or necrosis in the intestines, therefore a bridectomy with the removal of implant was performed. The postoperative course was uneventful and the patient was discharged on day five. A written consent form was also obtained from the patient for this study.

#### Discussion

The management of AIO is often a surgical challenge due to various clinical and radiological findings. It may be sometimes confused with other causes of acute abdomen, or the primary etiology may not be always clearly determined preoperatively, particularly in cases of incomplete obstruction. Similarly, our patient had physical findings of acute abdomen and a mild diarrhea with a minimal distension, which did not suggest an intestinal obstruction as initial diagnosis. Moreover, the ultrasound findings pointing perforated appendicitis, and nonspecific abdominal X-ray led to incorrect preoperative diagnosis.

It is well known that intra-abdominal adhesions are responsible for up to 70% of cases with AOI [5]. These adhesions are fibrous strands extending between various abdominal organs and structures, and are mostly associated with any intra-abdominal or pelvic surgery such as colectomy, appendectomy and hysterectomy [6]. Intra-abdominal infectious disorders and foreign bodies such as peritoneal dialysis catheters and drains can also cause strong adhesions within the abdominal cavity, which can lead to AIO. However, migration of a penile implant into the abdomen causing an intestinal obstruction is an exceptionally rare condition. In the literature, there are a few reports on this unusual complication of penile prosthetic devices, which are mostly as single cases. Penile prosthetic devices have been commonly used in patients with erectile dysfunction refractory to medical treatment over the past 50 years, and supplement the function of the erectile bodies to achieve penile rigidity, just like a real erection [7]. These devices can be either external or implanted. Despite the technological improvements in inflatable penile prosthesis in recent years, it still carries risk of complication including infection, mechanical problems, injury to bladder and surrounding tissues, herniation to the abdominal cavity causing intra-abdominal injuries or bowel obstruction [4,8]. Intestinal obstruction can be caused by the intraluminal migration of the reservoir of the penile prosthesis or fibrous strands between the herniated reservoir and intestinal loops. Bridectomy is a sufficient surgical intervention for the cases of bowel obstruction with mild operative findings, as was in our case. Resection of affected loops is required in case of bowel necrosis or perforation.

In conclusion, intestinal obstruction is a common surgical disorder, with various physical symptoms and signs from mild to severe. Therefore, the patients presenting with the clinical findings of intestinal obstruction or acute abdomen syndrome should be evaluated as a whole, especially in terms of their medical history. Although rare, AIO due to penile implant always should be kept in mind for an accurate management of such cases.

### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Cappell MS, Batke M. Mechanical obstruction of the small bowel and colon. Med Clin North Am 2008;92(3):575-97
- 2. Nelson RP Jr. Small bowel obstruction secondary to migration of an inflatable penile prosthesis reservoir: recognition and prevention. J Urol 1988;139(5):1053-
- 3. Luks FI, Huntley HN, Tula JC, Pizzi WF. Small-bowel obstruction by an inflatable penile prosthesis reservoir. Surgery 1989;106(1):101-4.

- 4. Serio SJ, Schafer P, Merchant AM. Incarcerated inguinal hernia and small bowel obstruction as a rare complication of a penile prosthesis. Hernia 2013;17(6):809-
- 5. Parker MC, Ellis H, Moran BJ, Thompson JN, Wilson MS, Menzies D, et al. Postoperative adhesions: ten-year follow-up of 12,584 patients undergoing lower abdominal surgery. Dis Colon Rectum 2001;44(6):822-9; discussion 829-30.
- 6. Catena F, Di Saverio S, Kelly MD, Biffl WL, Ansaloni L, Mandala V, et al. Bologna guidelines for diagnosis and management of adhesive small bowel obstruction (ASBO): 2010 evidence-based guidelines of the World Society of Emergency Surgery. World J Emerg Surg 2011;6:5.
- 7. Le B, Burnett AL. Evolution of penile prosthetic devices. Korean J Urol 2015;56(3):179-86.
- 8. Efesoy O, Özbay E, Tek M, Akbay E, Çayan S. Erektil disfonksiyon tedavisinde üç parçalı şişirilebilir penil protez cerrahisinin uzun dönem sonuçları ve çiftlerin memnuniyeti. Turkiye Klinikleri J Med Sci 2010;30(6):1965-70.

#### How to cite this article:

Baba S, Yıldırım O, Kılıç M.Ö, Hatipoğlu N.D, Güldoğan C,E. A Rare Case of Acute Intestinal Obstruction Due to the Migration of Penile Prosthesis. J Clin Anal Med 2015;6(suppl 5): 641-3.

## Is latrogenic Cushing's a New Form of Child Neglect?

## İyatrojenik Cushing Yeni Bir Çocuk İhmali Formu Olabilir Mi?

Yeni Bir Çocuk İhmali Formu / A New form of Child Neglect

Habip Almiş<sup>1</sup>, Ahmet Köse<sup>1</sup>, İbrahim Hakan Bucak<sup>1</sup>, Eda Mengen<sup>2</sup>, Mehmet Turgut<sup>1</sup>

<sup>1</sup>Department of Pediatrics, Adiyaman University, Faculty of Medicine, Adiyaman,

<sup>2</sup>Department of Pediatric Endocrinology, Cukurova University, Faculty of Medicine, Adana, Turkey

#### Ozet

Çocuk istismarı, giderek önem kazanan bir konu olup bir çocuk istismarı tipi olan çocuk ihmali; bilerek veya bilmeyerek çocuğa zarar verme davranışıdır. Bu yazımızda bez dermatiti nedeniyle 11 ay boyunca topikal klobetazol 17-propionat kullanılması sonucu iyatrojenik cushing sendromu gelişen on üç aylık bir erkek çocuk olgu, iyatrojenik Cushing sendromunun bir çeşit çocuk ihmali olup olmadığının tartışılması amacıyla sunulmaktadır. Çocuğunun sağlığını korumak ve hastalandığında tedavisi ile ilgili yapılması gerekenleri öğrenmek ailelerin temel görevlerinden biridir. Sağlık çalışanları tarafından ailelerin yeterli oranda bilgilendirilmesinin sağlık alanındaki çocuk ihmallerini azaltacağı kanısındayız.

### Anahtar Kelimeler

İyatrojenik Cushing; Çocuk İhmali; Kortikosteroit

#### Abstract

Child abuse is an increasingly important issue. One of the main types of abuse is child neglect, that is, behavior in which the child is knowingly or unknowingly injured. In this article; we report a case of iatrogenic Cushing's syndrome due to clobetasol 17-propionate treatment that was used inappropriately, in order to discuss whether or not this is a form of child neglect. It is one of the basic tasks of families to protect the health of the child and learn how to treat that child when he or she is ill. We believe that by being adequately informed by the health professionals, families may reduce the risk of child neglect.

### Keywords

latrogenic Cushing's; Child Neglect; Corticosteroid

DOI: 10.4328/JCAM.3727 Received: 02.07.2015 Accepted: 31.07.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 644-6 Corresponding Author: Habip Almis, Department of Pediatrics, Adiyaman University, Faculty of Medicine, 02040, Adiyaman, Turkey.
T.: +90 4162161015 E-Mail: drhabipalmis@yahoo.com

Diapers sometimes cause inflammation where they come into contact with the skin, a condition known as "diaper dermatitis". As it is generally improved with short-term topical corticosteroid therapy, topical corticosteroids are often the preferred group of drugs appropriate or inappropriate prescribed by doctors to treat this condition [1].

Corticosteroids are generally effective when used at the appropriate dosage and for the appropriate duration in accordance with the patient's age and symptoms; however, quite serious systemic and local side effects may appear if used in inappropriate doses for a long period of time [2,3].

Child abuse is an increasingly important issue. One of the main types of abuse is child neglect, that is, behavior in which the child is knowingly or unknowingly injured.

In this article; we report a case of iatrogenic Cushing's syndrome due to clobetasol 17-propionate treatment that was used inappropriately, in order to discuss whether or not this is a form of child neglect.

### Case Report

A thirteen-month-old male patient was admitted to our clinic with a cough and fever. Our patient was born at term weighing 3750 grams and was the 31-year-old mother's third pregnancy. Due to diaper dermatitis, the infant was prescribed clobetasol 17-propionate at the age of two months. The dermatitis did not improve, so the family continued using the same drugs on the area for eleven months.

On physical examination, pulse rate was 92 beats / min, respiration was 27 breaths / min, blood pressure was 80/50 mmHg, body weight was 8.8 kg (10-25 percentile), and height was 76 cm (10-25 percentile). The patient had a Cushingoid appearance (Figure 1). Laboratory findings showed that the complete blood count and biochemical values were within normal limits. When the patients' blood was taken in the morning, the level of adrenocorticotropic hormone (ACTH) was 7.22 pg / ml (normal range 10-50 pg / ml), and cortisol levels <0.01 mg / dl (normal range 6.7 to 22.6 mg / dL).

latrogenic Cushing's syndrome was diagnosed in the patient, and use of the cream used was immediately ceased. The Cushingoid appearance of the patient was also seen to improve on subsequent follow-up.

#### Discussion

Child abuse and neglect are serious problems worldwide, having lasting negative impacts on the physical and mental health of children. Forms of child abuse include physical abuse, sexual abuse, emotional abuse, and child neglect. There are many different subtypes of child neglect. Physical neglect is the failure to provide appropriate nutrition, hygiene, clothing, supervision, and medical care for children, while medical neglect can occur in different forms, and can cause harm or death in some cases [4-6].

Many factors can lead to medical neglect, preventing children from receiving appropriate medical care. Caregivers may not recognize signs or symptoms in their children that could be a precursor to serious illness. They may also understand why it is important to follow the instructions of the physician [7,8].



Figure 1. Cushingoid appearance of patient

Long-term use of topical corticosteroids that caused iatrogenic Cushing's syndrome in our patient could count as a form of medical neglect, which emerged due to the ignorance and low educational level of the family. Families should have sufficient knowledge to protect their children's health. If they do not seek treatments for diseases when they occur, this is a form of passive child neglect. We believe that it is essential that the information provided by health professionals is clearly expressed so that families can understand how to use prescribed drugs and administer treatment.

It is one of the basic tasks of families to protect the health of the child and learn how to treat that child when he or she is ill. We believe that by being adequately informed by the health professionals, families may reduce the risk of child neglect.

### Competing interests

The authors declare that they have no competing interests.

1. Gokalp AS, Aldirmaz C, Oguz A, Gultekin A, Bakıcı MZ. Relation between the intestinal flora and diaper dermatitis in infancy. Trop Geogr Med 1990;42(3):238-

- 2. Hughes J, Rustin M. Corticosteroids. Clin Dermatol 1997;15:715–21.
- 3. Keane FM, Munn SE, Taylor NF, du Vivier AW. Unregulated use of clobetasol propionate. Br J Dermatol 2001;144:1095-6.
- 4. Young JC, Widom CS. Long-term effects of child abuse and neglect on emotion processing in adulthood. Child Abuse Negl 2014;38:1369–81.
- 5. Habetha S, Bleich S, Weidenhammer J, Fegert JM. A prevalence-based approach to societal costs occurring in consequence of child abuse and neglect. Child Adolesc Psychiatry Ment Health 2012;6:35.
- 6. Crawford M. Physical abuse: pitfalls and challenges. Paediatrics and Child Health 2010;20:566-70.
- 7. Dubowitz H. Neglect of children's health care. In: Myers JEB, Berliner L, Briere J, Jenny C, Hendrix CT, Reid T, editors. The APSAC Handbook on Child Maltreatment. 2nd ed. Thousand Oaks CA: Sage Publications; 2002. p.269-92.
- 8. Jenny C. Recognizing and responding to medical neglect. Pediatrics 2007;120(6):1385-9.

### How to cite this article:

Almiş H, Köse A, Bucak İ.H, Mengen E, Turgut M. Is latrogenic Cushing's a New Form of Child Neglect? J Clin Anal Med 2015;6(suppl 5): 644-6.

### **Breast Cancer with Silent Metastasis to Uterine Cervix**

Meme Kanseri'nin Serviks Uteri Metastazı / Cervical Metastasis of Breast Cancer

Gokhan Pekindil<sup>1</sup>, Pinar Solmaz Hasdemir<sup>2</sup>, Ayca Tan<sup>3</sup>, Gamze Goksel<sup>4</sup> <sup>1</sup>Department of Radiology, <sup>2</sup>Department of Obstetrics and Gynecology, <sup>3</sup>Department of Pathology, <sup>4</sup>Department of Medical Oncology, Celal Bayar University School of Medicine, Manisa, Turkey

Kadın genital organlarına ekstragenital kanserlerin metastaz yapmasına nadiren rastlanır. Uterin serviks, kadın genital sisteminde en nadir olarak dış organ metastazı alan bölgedir. Bu yazıda, overler ve uterin servikse sessiz metastaz yapmış bir opere meme kanseri olgusu sunulmuştur. Öyküsünde meme kanseri olan olgularda jinekolojik organların metastaz yönünden dikkatlice değerlendirilmesi, özellikle vajinal kanama veya şüpheli ultrasonografik bulguları olan olgularda olmak üzere önem taşımaktadır. Uterin serviks de potansiyel bir metastatik saha olarak mutlaka değerlendirme kapsamına alınmalıdır.

#### Anahtar Kelimeler

Meme Kanseri; Metastaz; Serviks; Ovaryan Metastaz

Metastases to female genital organs from extragenital cancers are uncommon. Uterine cervix is one of the less uncommon site of the genital tract for extragenital organ tumors. We present a case of operated breast cancer who had silent metastases to the ovaries and uterine cervix. Metastatic involvement of the gynecologic organs should be considered in women with a history of breast cancer who present with vaginal bleeding or suspicious changes on transvaginal ultrasound. Uterine cervix should always kept in mind as a part of metastasis.

### Keywords

Breast Cancer; Cervix; Metastasis; Ovarian Metastasis

DOI: 10.4328/JCAM.3748 Corresponding Author: Pinar Solmaz Hasdemir, Department of Obstetrics & Gynecology, Celal Bayar University School of Medicine, 45000, Manisa, Turkey. T.: +90 4444228 F.: +90 2362338040 E-Mail: solmazyildiz@yahoo.com

Metastasis of distant malignancies to the cervix uteri is a rare occurrence with a frequency of 0,3 % for all tumors [1,2]. However, the frequency of cervical metastasis of breast cancer is much lower and there are only 35 breast cancer with cervical metastasis cases reported in literature [3]. Because of the situation was rather uncommon, it might be difficult to diagnose for the clinician.

#### Case Report

A 40 years old woman with a history of breast cancer was referred to the gynecology out-patient clinic for routine control. She had a history of left sided total mastectomy and axilllary lymph node dissection operation because of a malign mass in her left breast and 6 courses of TAC (Docetaxel, Adriamycin, Cyclophosphamide) chemotherapy and 25 days of radiotherapy (a total dose of 5000 cGy) after the operation. She was at the 20th months of the adjuvant chemotherapy. Hormonotherapy with Goserelin and Tamoxifen was planned because of the positive (90%) estrogen and progesterone hormone receptors in the pathologic examination of the tumoral tissue. The physical examination and findings at imaging of the genital organs were completely normal (Figure 1). Total hysterectomy with

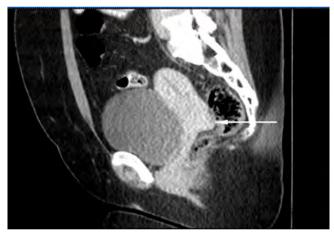


Figure 1. No finding is seen in servix (arrowed) on sagitally reformatted pelvic MDCT.

bilateral adnexectomy was planned because of the patient's anxiety about the breast tumor and willingness for the operation. The appearance of the intraabdominal organs including the uterus and the adnexes were normal. The pathologic investigation revealed that metastasis of invazive lobular breast cancer in bilateral ovaries and the uterine cervix. The microscopic tumor areas was found in the exocervical area and in the lymphatic vessels. On macroscopic examination there was not any mass in the uterine cervix, uterus and bilateral ovaries. Endometrium and myometrium had no significant pathological findings. However, beneath the normal squamous epithelium of the cervix and in more deeper stromal areas there were multiple foci of non-cohesive cells having hyperchromatic nuclei and scanty cytoplasm, some with signet ring-cell appearance. These cells formed no organoid pattern (Figure 2). In some areas the tumor cells were seen in lymphatics forming thrombi. On sections from both ovaries, similar cells were found to diffusely infiltrated ovarian stroma (Figure 3). Immunohistochemical (IHC) analysis revealed reactivity for cytokeratin, estrogen

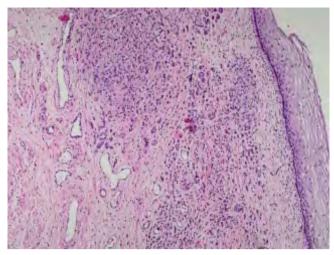


Figure 2. Foci of tumor comprising with non-cohesive cells just beneath the squamous epithelium of the uterine cervix (H&EX100).

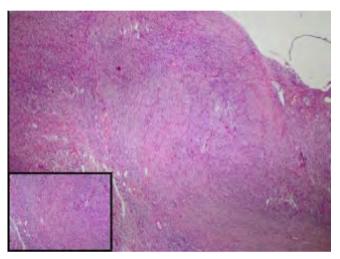


Figure 3. Diffusely infiltrating tumor cells of in the ovary (H&EX40), inset

receptor (ER), progesterone receptor (PR), mammoglobulin and gross cystic disease fluid protein-15 (GCDFP-15), whereas the cells were negative for E-cadherin (Figure 4-5). All these findings were consistent with the patient's history having lobular carcinoma in her breast . The result of the pathologic investigation was consulted with medical oncology department and additional chemotherapy with Gemcitabine and Kapesitabin (1-8/28 days) was decided to start. The patient is under the 4th course of chemotherapy with normal imaging on computerized tomographic examination.

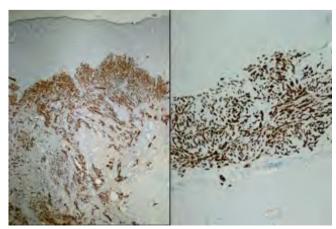


Figure 4. Positivity of tumor cells with cytokeratin, left side-ovary (X40) and right side-uterine cervix (X100).

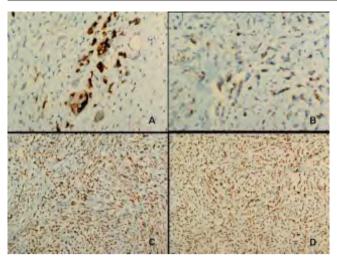


Figure 5. Tumor cells infiltrating uterine cervix showing positivity with A) mammoglobulin (X400) B) GCDFP-15 (X400) C) PR (X200) D) ER (X200).

#### Discussion

Uterine cervix is a curious area for extragenital tumor metastases. Mazur et al collected a series of 149 cases metastases to the female genital tract from extragenital cancers and only 3,4% (5/149) cases were found to be metastasized to uterine cervix [4]. The most common sites of the primary tumor were the breast and the stomach in cervical metastasis from the extragenital organ cases [5].

Although genital organs are not common sites for the metastatic spread of the extragenital tumours, if a metastatic spread is being discussed, the ovaries are the most common gates to the genital tissues because of the intrinsic features of ovarian stroma. It is thought that, the uterus and cervix might be possibly influenced by lymphatic spread from the ovary to the other genital sites [3].

The most common presenting symptom was abnormal vaginal bleeding (57%) followed by suspicious changes of the cervix on transvaginal ultrasound [6]. However, no clinical sign was present in 32% of the patients in literature and 41% of the reported cases were found only at autopsy [7]. Cervical cytology could diagnosed the neoplastic cells in only 50% of these cases [1]. No gynecologic sign or symptom was found in our patient and the result of the cervical cytologic was clear, as well. The only indication for operation was patient's anxiety and persistence for the operation. Prophylactic gynecologic operations in patients with predisposition to cancer are shown to be reduced the overall cancer risk. However, early onset menopause is an adverse effect which had to be discussed with the patient [8]. Metastatic involvement of the genital organs including the cervix should be considered in women with a history of breast cancer who present with vaginal bleeding or abnormal ultrasound. It should be kept in mind that sometimes these metastases might be asymptomatic and patient's desires should be taken into account while giving decision about her disease.

### Competing interests

The authors declare that they have no competing interests.

1. Pérez-Montiel D, Serrano-Olvera A, Salazar LC, Cetina-Pérez L, Candelaria M, Coronel J, et al. Adenocarcinoma metastatic to the uterine cervix: a case series. J Obstet Gynaecol Res 2012;38(3):541-9.

- 2. Abrams HL, Spiro R, Goldstein N. Metastases in carcinoma: analysis of 1000 autopsied cases. Cancer 1950;3(1):74-85.
- 3. Bogliolo S, Morotti M, Valenzano Menada M, Fulcheri E, Musizzano Y, Casabona F. Breast cancer with synchronous massive metastasis in the uterine cervix: a case report and review of the literature. Arch Gynecol Obstet 2010;281(4):769-73. 4. Mazur MT, Hsueh S, Gersell DJ. Metastases to the female genital tract: analysis of 325 cases. Cancer 1984;53(9):1978-84.
- 5. Piura B, Yanai-Inbar I, Rabinovich A, Zalmanov S, Goldstein J. Abnormal uterine bleeding as a presenting sign of metastases to the uterine corpus, cervix and vagina in a breast cancer patient on tamoxifen therapy. Eur J Obstet Gynecol Reprod Biol 1999:83(1):57-61.
- 6. Famoriyo A, Sawant S, Banfield PJ. Abnormal uterine bleeding as a presentation of metastatic breast disease in a patient with advanced breast cancer on tamoxifen therapy. Arch Gynecol Obstet 2004;270(3):192-3.
- 7. Hepp HH, Hoos A, Leppien G, Wallwiener D. Breast cancer metastatic to the uterine cervix: analysis of a rare event. Cancer Invest 1999;17(7):468-73.
- 8. Rosen B, Kwon J, Fung Kee Fung M, Gagliardi A, Chambers A. Systematic review of management options for women with a hereditary predisposition to ovarian cancer. Gynecol Oncol 2004;93(2):280-6.

#### How to cite this article:

Pekindil G, Solmaz Hasdemir P, Tan A, Goksel G. Breast Cancer with Silent Metastasis to Uterine Cervix. J Clin Anal Med 2015;6(suppl 5): 647-9.



# Co-existance of Lymph Node Tuberculosis and Pulmonary Embolism: A Case Report

# Lenf bezi Tüberkülozu ve Pulmoner Emboli Birlikteliği: Olgu sunumu

Lenf bezi Tüberkülozu ve Pulmoner Emboli Birlikteliği / Lymph Node Tuberculosis and Pulmonary Embolism

İbrahim Koç<sup>1</sup>, Serdar Doğan<sup>2</sup>, Yusuf Doğan<sup>3</sup>, Ahmet Ulusan<sup>4</sup>

<sup>1</sup>Pulmonary Medicine, Viransehir State Hospital, Sanliurfa, <sup>2</sup>Biochemistry, Viransehir State Hospital, Sanliurfa,

<sup>3</sup>Microbiology, Viransehir State Hospital, Sanliurfa, <sup>4</sup>Thoracic Surgery, Hitit University Corum Education and Research Hospital, Corum, Turkey

#### Ozet

Pulmoner emboli vücudun başka bir yerinden kaynaklanan bir oluşumla ile pulmoner arterlerin tıkanması ve erken teşhis ve tedavi edilmediği takdirde yüksek ölüm oranına sahip bir hastalıktır. Tüberküloz Mycobacterium tuberculosis'in neden olduğu genellikle akciğerleri etkileyen bir hastalık olup vücudun diğer organları da etkilenebilmektedir. Burada nefes darlığı, kilo kaybı ve gece terlemeleri, nefes darlığı şikayetleri ile kliniğimize başvuran altmış üç yaşında bir kadın hasta sunulmaktadır. İleri yaşta kilo kaybı ve gece terlemesi, bir maligniteyi düşündürmüş olmakla beraber tümör belirteçleri negatif saptandı. Sigara içmeyen daha önceden sağlıklı olan bir bireyde düşük oksijen saturasyonu pulmoner emboliden şüphelendirmişti olan bir bireyde düşük oksijen saturasyonun peliren en en boli saptandı. Servikal lenf nodu eksizyonunun patolojik değerlendirmesinde kazeifikasyon nekrozu saptandı. Sonuç olarak pulmoner emboli tanılı olan hastalarda kilo kaybı ve oksijen saturasyonunda düşme varsa maligniteyle beraber tüberkülozun da ekarte edilmesi gerekmektedir.

#### Anahtar Kelimeler

Pulmoner Emboli; Lenf Bezi; Tüberküloz

#### Abstract

Pulmonary embolism is occlusion of pulmonary arteries with a material originating from another part of the body and has a high fatality rate if not diagnosed and managed early. Tuberculosis is an infection caused by mycobacterium tuberculosis, generally effecting lungs but involvement of other parts of the body is possible. Here we report a sixty three years old woman who admitted to our clinic with complaints of shortness of breath, weight loss and night sweats. Weight loss and night sweats in old age were suggestive of a malignancy but tumor markers were negative. Low oxygen saturation in a non-smoking previously healty person arise suspicion of pulmonary embolism. Computed tomography pulmonary angiography revealed lymphadenopathy and pulmonary embolism. Pathology of the servical lymph node revealed caseation necrosis. In conclusion in patients with pulmonary embolism who has weight loss and low oxygen saturation beside the malignancy tuberculosis also should be excluded.

#### Keywords

Pulmonary Embolism; Lymph Node; Tuberculosis

DOI: 10.4328/JCAM.3758 Received: 14.07.2015 Accepted: 31.07.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 650-2 Corresponding Author: Ibrahim Koc, Pulmonary Medicine, Viransehir State Hospital, Sanliurfa, Turkey.
GSM: +905453142502 E-Mail: İbrahimkoc1981@gmail.com

Pulmonary embolism (PE) is occlusion of pulmonary arteries with a material originating from another part of the body and has a high fatality rate if not diagnosed and managed early. The causes of pulmonary embolism can be venous thromboembolism (VTE), and nonthrombotic embolism like septic, fat, air, amniotic fluid and tumour embolism. Patients might present with different variable clinical presentation and often have nonspecific complaints which make the diagnosis challenging. The reported annual incidence of VTE differs ranging between 23 and 69 cases per 100,000 population, [1, 2] with approximately one third of patients presenting with acute PE and two thirds with deep vein thrombosis [3]. Tuberculosis is a major health problem in undeveloped and developing countries like Turkey with a various presentations and complications. Tuberculosis can lead to hypercoagulability, increased venous stasis, and endothelial dysfunction, thus increasing the susceptibility to (VTE) and pulmonary thrombo embolism (PTE). Here we report a sixty three years old woman who had lymph node tuberculosis and PE.

#### Case Report

A sixty three years old woman admitted to our clinic with complaints of shortness of breath, weight loss and night sweats. Her medical and family histories were unremarkable. Her physical examination revealed normal body temperature (36oC), heart rate of 100 beats per minute, respiratory rate of 20 breaths per minute, blood pressure of 110/80 mm Hg, and SpO2 of 85% on room air. Physical examination revealed servical lymphadenopathy (Picture 1). Physical examination of the chest revealed



Picture 1. Enlarged servical lymph nodes of the patient.

normal vesicular sounds. Weight loss and night sweats in old age were suggestive of a malignancy but tumor markers were negative. Low oxygen saturation in a non-smoking previously healty person arise suspicion of pulmonary embolism. Computed tomography pulmonary angiography revealed lymphadenopathy and pulmonary embolism (Figure 1). Servical lymph node excision revealed caseation necrosis (Picture 2).

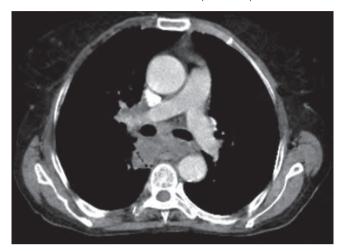
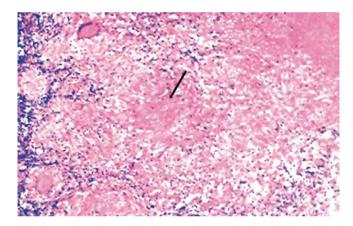


Figure 1. Mediastinal lymphadenopathy and pulmonary embolism.



Picture 2. Caseation necrosis of the lymph node specimen (Black arrow) (HE×200).

#### Discussion

Tuberculosis is a major health and public problem in undeveloped and developing countries. PTE is the third greatest cause of mortality from cardiovascular disease, after myocardial infarction and cerebrovascular stroke [4]. The real number is likely to be much more than expected, since the condition goes unrecognised in many patients. In a study Ozbay et al. reported a higher rate of pleural and meningeal involvement among extrapulmonary tuberculosis cases than expected [5]. Drawing attention to tuberculous meningitis because of its high mortality rate. Tuberculosis mostly effects the lungs but may have more serious outcomes like meningitis and pulmonary embolism. This case highlights the occurrence of lymph node tuberculosis and pulmonary embolism in a patient with lymph node tuberculosis with no risk factors for thromboembolism, a significant but rare association posing a diagnostic dilemma which may have serious prognostic implications.

It has been shown that recent respiratory infection, acute infection and raised inflammatory markers, are associated with

increased risk of thromboembolic disease [6]. It has been reported that prevelance of PTE in patients with pneumonia as high as 10% [7]. Another study has proven that there are strong associations between recent respiratory infection and venous thromboembolism which may be related to the severity of the infection [6]. There are obvious evidences about the hypercoagulability state in tuberculosis [8]. Co-existance of lymph node tuberculosis and pulmonary embolism is a rare entity. Increase in plasma fibrinogen and factor VIII, and reactive thrombocytosis might be reasons of hypercoagulability in tuberculosis patients. In our case, in addition to above mentioned factors stasis due to local compression of veins by the enlarged reactive lymph nodes or immobility caused by respiratory compromise, and or endothelial dysfunction might be reasons leading to pulmonary embolism.

In conclusion in patients with pulmonary embolism who has weight loss and low oxygen saturation beside the malignancy tuberculosis also should be excluded. Although the differential diagnosis is difficult in tuberculosis because of the non-spesific clinical and radiological findings, still is easier than of pulmonary embolism which requires specific diagnostic tools. Clinicians must be carefull, acute onset of symptoms like shortness of breath, pleuritic chest pain, and hypoxemia must arise suspician.

### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Silverstein MD, Heit JA, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ, 3rd. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25year population-based study. Arch Intern Med 1998;158(6):585-93.
- 2. Anderson FA, Jr., Wheeler HB, Goldberg RJ, Hosmer DW, Patwardhan NA, Jovanovic B, et al. A population-based perspective of the hospital incidence and casefatality rates of deep vein thrombosis and pulmonary embolism. The Worcester DVT Study. Arch Intern Med 1991;151(5):933-8.
- 3. White RH. The epidemiology of venous thromboembolism. Circulation 2003;107(23 Suppl 1):I4-8.
- 4. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999:353(9162):1386-9.
- Ozbay B, Uzun K. Extrapulmonary tuberculosis in high prevalence of tuberculosis and low prevalence of HIV. Clin Chest Med 2002;23(2):351-4.
- 6. Clayton TC, Gaskin M, Meade TW. Recent respiratory infection and risk of venous thromboembolism: case-control study through a general practice database. Int J Epidemiol 2011;40(3):819-27.
- 7. Rohacek M, Szucs-Farkas Z, Pfortmuller CA, Zimmermann H, Exadaktylos A. Acute cardiac disorder or pneumonia and concomitant presence of pulmonary embolism. PLoS One 2012;7(10):e47418. PubMed PMID: 23091623. Epub 2012/10/24. eng.
- 8. Sharif-Kashani B. Bikdeli B. Moradi A. Tabarsi P. Chitsaz E. Shemirani S. et al. Coexisting venous thromboembolism in patients with tuberculosis. Thromb Res 2010;125(5):478-80.

### How to cite this article:

Koç İ, Doğan S, Doğan Y, Ulusan A. Co-existance of Lymph Node Tuberculosis and Pulmonary Embolism: A Case Report. J Clin Anal Med 2015;6(suppl 5): 650-2.



# Bladder Rupture and Urine Fistula Between-Bladder and Supracondylary Pin Tract After Pelvis Fracture: A Case Report

Pelvis Kiriği İle Birlikte Supra Kondiler İdrar Fistülü / Pelvic Fractures with Urine Supracondiler Fistula

Zekeriya Oztemur<sup>1</sup>, Umut Hatay Gölge<sup>2</sup>, Ömer Pıçakçı<sup>1</sup>, Gündüz Tezeren<sup>1</sup>, Okay Bulut<sup>1</sup> <sup>1</sup>Department of Orthopaedic and Traumatology, Cumhuriyet University School of Medicine, Sivas, <sup>2</sup>Department of Orthopaedics and Traumatology, Çanakkale Onsekiz Mart University School of Medicine, Çanakkale, Turkey

> 12. Ulusal Türk Ortopedi ve Travmatoloji Kongresinde poster sunumu yapılmıştır. [AOTT(Acta orthopaedica et tarumatologica turcica) cilt(vol.) 45 Suppl.1, Kasım (november) 2011]

Pelvik kırıklarının en önemli komplikasyonlarından biri mesane rüptürüdür. Pelvis kırıklarının potansiyel komplikasyonlarından biri de mesane yaralanmalarına bağlı olarak gelişen vesikovajinal,vesikorektal, vesikouterin fistüllerdir. Literatürde nadir rastlanan mesane rüptürü ile birlikte, femur suprakondiler bölgede çivi dibinden üriner fistülizasyon olgusunu sunmayı amaçladık. Pelvis kırığı olan hastalarda iskelet traksiyonu çivisi dibinden gelen sıvının tel dibi infeksiyonunun yanı sıra çok nadir bir komplikasyon olan mesane fistülizasyonu da olabileceği akılda tutulmalıdır.

#### Anahtar Kelimeler

Pelvis Kırığı; Mesane Rüptürü; Çivi Dibi; İdrar Fistülü

#### Abstract

One of the most important complications of pelvic fractures is bladder rupture. Potential complications of pelvic fractures caused by bladder ruptures are vesicovaginal, vesicorectal, vesicouterine and urethrorectal fistulas. Along with bladder rupture, which is rarely encountered in the literature, the case of urinary fistula from pin tract in femur supracondylar region was represented. The case of the bladder rupture induced by pelvic fracture that associated with urinary fistulisation between bladder and pin tract from femur supracondylar region was reported.

#### Keywords

Pelvis Fracture; Bladder Rupture; Pin Tract; Urine Fistula

DOI: 10.4328/JCAM.3542 Corresponding Author: Zekeriya Oztemur, Department of Orthopaedic and Traumatology, Cumhuriyet University School of Medicine, Sivas, Turkey. GSM: +905052394436 E-Mail: oztemurz@gmail.com

Roughly 90% of all bladder ruptures are associated with pelvic disruption; however, only 9% to 16% of all pelvic disruptions have a concomitant bladder rupture [1]. Vesicovaginal, vesicorectal, vesicouterine fistula is a well-known surgical problem defined as an abnormal connection between the urinary bladder, the vagina, the rectum and the uterine [1-4]. Vesicocutaneous fistulas are characterized as an aberrant connection between the urinary bladder and the skin. Their formation has been associated with postsurgical complications, trauma as the result of bladder entrapment by external pelvic fixator [5]. Furthermore, no report has been published describing bladder rupture and urine fistula between-bladder and supracondylar pin tract after pelvis fracture. We present the unique case of bladder rupture and urine fistula between-bladder and supracondylar pin tract after blunt pelvic trauma resulting from a traffic accident.

#### Case Report

The case is a 63 year-old female patient. As a result of in-vehicle traffic accident, in another centre pelvic external fixator and bilateral skeletal traction were applied to the case due to fracture of left ischium, right iliac wing fracture and right sacroiliac joint separation (Tile Type C1-2) [6] (Fig.1) . The case was transferred to emergency service of our hospital and primary



Figure 1. Left ischium pubic branch fracture, right iliac wing fracture, right sacroiliac joint separation (Tile Type C1-2) are seen in X-Ray graph.

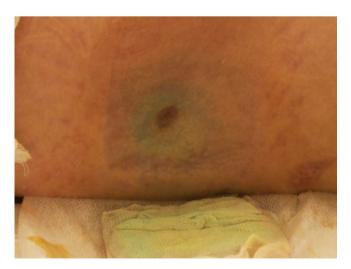


Figure 2. Leakage of methylene blue from pin tract after the bladder was filled with methylene blue



Figure 3. Leaking of radio opaque substance from bladder to pin tract according to bladder cystogram.

bladder repair was applied. Serous flow occurred in the pin tract in supracondylar region in 12th day of bladder repair. There were no bacteria or increase of polymorphonuclear leukocytes related pin tract infection in direct microscopic examination and aerobic culture taken from pin tract. Leakage of methylene blue was observed from the pin tract in supracondylar region after filling the bladder with methylene blue and a fistula between bladder (Fig. 2) and supracondylar pin tract was detected in bladder cystogram (Fig. 3). Bladder was repaired again.

#### Discussion

Since pin tract infection is the most significant complication associated with the use of external fixation, it has been reported to occur up to 63% of pins[6-8] . This high infection rate has been attributed to the conduit that the pins provide between the skin and underlying soft tissue and bone. Complications related to pin tract infection include a need for pin change or removal, failure of fracture healing, septic arthritis, and osteomyelitis. Therefore, a method to decrease the rate of pin infection has tremendous clinical appeal [6]. However, main macroscopic and microscopic main characteristics of reported case were not complied with the infection. Fluid is more serious and has a homogeneous colour. Another possible reason is that synovial fluid leaking from a pin placed near the joint. As a result of radiological controls, it was verified that there was no relationship between pin and joint. By virtue of cystogram and filling bladder with methylene blue, it was determined that the fluid was urine. Alexander and Sagi [9] reported a case having septic cox arthritis secondary to development of a cystosynovial fistula after non-operative treatment for both a pelvic fracture and bladder rupture. Tolkach et al.[10] reported Vesico-Acetabular Fistula and Urolithiasis in the hip joint cavity due to persistent bladder entrapment after acetabular fracture. However, there has been no similar case in the literature.

In conclusion, it should be remembered that in patients having pelvic fracture along with bladder and urethral injury, fistula between bladder and pin tract can occur as well as possible reasons such as pin tract infection or synovial fluid leakage in case of serous fluid from pin tract.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Brandes S, Borrelli Jr J. Pelvic fracture and associated urologic injuries. World J Surg 2001;25(12):1578-87.
- 2. Kalorin CM, Rosati C, Markarian M. Rectovesical Fistula in Association with Vesicocutaneous Fistula After Blunt Pelvic Trauma. J Trauma 2008;65(4):34-5.
- 3. Kochakarn W, Ratana-Olarn K, Viseshsindh V, Muangman V, Gojaseni P. Vesicovaginal fistula: experience of 230 cases. J Med Assoc Thai 2000;83(10):1129-32.
- 4. Benchekroun A, Lachkar A, Soumana A, Farih MH, Belahnech Z, Marzouk M, et
- al. Urethrorectal fistula. Report of 11 cases. Ann Urol (Paris) 1999;33(2):93-6. 5. Chitale SV, Almeida A, Hadfield JI. Urological complication of pelvic fracture treated by an external fixator. Br J Urol 1997;80(4):681-2.
- 6. Tile M. Acute pelvic fractures: I. Causation and classification. J Am Acad Orthop Surg 1996 ;4(3):143-51.
- 7. Mahan J, Seligson D, Henry S, Hynes P, Dobbins J. Factors in pin tract infections. Orthopedics 1991;14(3):305-8.
- 8. Coester LM, Nepola JV, Allen J, Marsh JL. The effects of silver coated external fixation pins. lowa Orthop J 2006;26:48-53.
- 9. Alexander G, Sagi HC. Septic Arthritis of the Hip After Nonoperative Treatment of a Pelvic Fracture Associated With Bladder Rupture. J Orthop Trauma 2012;26(5):40-2.
- 10. Tolkach Y, Gadjiev N, Korol V, Gonchar I. Vesico-acetabular fistula and urolithiasis in the hip joint cavity due to persistent bladder entrapment after acetabular fracture.Korean J Urol 2011;52(3):221-4.

#### How to cite this article:

Oztemur Z, Gölge U.H, Pıçakçı Ö, Tezeren G, Bulut O. Bladder Rupture and Urine Fistula Between-Bladder and Supracondylary Pin Tract After Pelvis Fracture: A Case Report. J Clin Anal Med 2015;6(suppl 5): 653-5.



# Importance of CT Imaging on Spontaneous Rupture of Renal Angiomyolipoma: A Case Report

# Renal Anjiyomiyolipomların Spontan Rüptüründe BT Görüntülemenin Önemi: Olgu Sunumu

Rüptüre Renal Anjiyomiyolipom / Ruptured Renal Angiomyolipoma

Fatma Gundogdu¹, Aylin Okur¹, Halil İbrahim Serin¹, Sebahattin Albayrak²¹Department of Radiology, ²Department of Urology, Bozok University Medical Faculty, Yozgat, Turkey

#### Özet

Renal anjiomiyolipomların çapı arttıkça anevrizma gelişme ve rüptüre olma riski artmaktadır. Yaşamı tehdit eden intratümoral kanama gibi ciddi komplikasyonlar gelişebilir. Kanamaları durdurmal için acil girişim gerekebilir. Bu yazıda, anjiyomiyolipomun spontan rüptürü sebebiyle retroperitoneal kanaması olan 16 yaşındaki kız hastanın tanısında bilgisayarlı tomografinin önemini sunmayı amaçladık.

### Anahtar Kelimeler

Anjiyomiyolipoma; Anevrizma; Rüptür; Tomografi

#### Abstract

Renal angiomyolipomas have a high risk of rupture when they are large and associated with aneurysms. The most serious complication that may occur is lifethreatening intratumoral bleeding. Immediate interventional therapies to stop bleeding are required. Herein, we report on a 16-year-old female patient with a retroperitoneal hematoma due to a spontaneous renal angiomyolipoma rupture by computed tomography.

### Keywords

Angiomyolipoma; Aneurysm; Rupture; Tomography

DOI: 10.4328/JCAM.3697 Received: 25.06.2015 Accepted: 06.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 656-8 Corresponding Author: Aylin Okur, Department of Radiology, Bozok University Medical Faculty, Yozgat, Turkey. GSM: +905334910575 E-Mail: draylinokur@hotmail.com

Angiomyolipoma (AML) is a benign hamartomatous tumor consisting of abnormal fat, muscle and vascular elements. AML is most often asymptomatic and incidentally we encounter in routine examination. However, sometimes it may cause symptoms such as recurrent hematuria or pain. Early diagnosis is crucial. Becauset he most serious complication that may occur is lifethreatening intraintratumoral bleeding [1].

There are two known types of AML: the isolated angiomyolipoma, which represents 80% of cases, and the one associated with tuberous sclerosis (TS). When associated with TS are often multiple, bilateral and small, and hemorrhage is frequent [2]. The MDCT is a gold standard in the diagnosis and complications of AML, whether or not clinical symptoms.

#### Case Report

A 16-year-old female was admitted with sudden onset of severe left flank pain. She had been diagnosed to have bilateral AML with tuberous sclerosis 5 years earlier.

The non-contrast computed tomography (CT), 7 mm hypodensity multipl nodular lesions in the bilateral kidneys were detected (Fig. 1A,B). However, in contrast enhanced multidetector

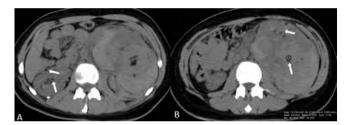


Fig.1. Non-enhanced CT scans at the bilateral kidneys show a low-density small lesions associated with a few interior fat components (arrows) and a hemorrhage presented spread into the left perirenal space (A,B).

computed tomography (MDCT) showed heterogeneous lesion of maximum diameter of 74x98 mm of the left kidney expanding into the perirenal space associated with a perinephric hematoma. The MDCT revealed inhomogeneous enhancement of the lesion and the presence of a few aneurysms ranging from 6 to 18 mm within the tumor (Fig. 2A,B). The lesion was diag-

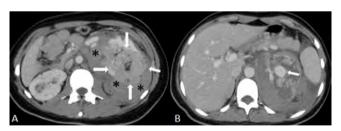


Fig.2. In contrast enhanced multidetector computed tomography (MDCT) showed heterogeneous lesion of maximum diameter of 74x98 mm of the left kidney (arrows) expanding into the perirenal space associated with a perinephric hematoma (asterisks) (A) and a few aneurysms presented within the tumor (arrow) (B)

nosed as an AML that had ruptured into the perinephric space. The patient has referred to Department of Interventional Radiology for transcatheter arterial embolization. Follow-up US performed 5 months after embolization showed significant tumor regression and the disappearance of perinephric hematoma.

#### Discussion

Angiomyolipoma is a benign hamartomatous tumor consisting of abnormal fat, muscle and vascular elements. AMLs usually arise from the renal cortex and tend to have an exophytic growth pattern, and when ruptured they cause perirenal hematoma to form as in our case. If AMLs will be grow and increase blood flow entering, they are likely to form aneurysms due to abnormal elastin-poor vascular structures [3]. For this reason, spontaneous rupture and induce hemorrhage possibility are quite high. The most reliable predictors of rupture are tumor size and aneurysm formation. If the tumor is larger than 4 cm and is associated with aneurysms larger than 5 mm, AMLs have a high risk of rupture [4]. The another risk factors associated with the spontaneous rupture and perirenal or intratumoral bleeding, include: association with tuberous sclerosis, signs and symptoms, a and pregnancy [5]. In our case, CT performed a lesion of maximum diameter of 74x98 mm and she had been diagnosed to tuberous sclerosis 5 years earlier.

The hemorrhage is usually limited to the perirenal space (PS) but, in some cases, may spread beyond the PS and involve the other retroperitoneal fasciae and fascial spaces. Hemorrhages that occur suddenly in association with acute shock can be lifethreatening, and their clinical manifestations are easily misun-

Ultrasound (US) has allowed diagnosing it without the need for biopsy in the majority of cases. The tumor presents a hyperechoic mass with great vascularity— existence of arteriovenous shunts on US /Doppler US (3). US scan may always identify the small aneurysms in the tumour. Hence, CT is the gold standard in the diagnosis of AML because it detects fat in the tumorhighly suggestive of AML. MDCT provides multiphasic and/ or 3D images with an advantage of shorter scanning times, thinner slice thickness and shows aneurysm formations. Magnetic Resonance imaging (MRI) can also differentiate fat by its high signal intensity and is a helpful tool exam currently used when CT is contraindicated [5]. In the present case, contrastenhanced MDCT revealed the location of aneurysms relevant to perinephric hemorrhage.

In symptomatic cases or with bilateral lesions, the choice should be selective arterial embolization or conservative renal surgery such as lumpectomy or partial nephrectomy [7]. Radical nephrectomy has been a valuable treatment strategy in cases of urgency, with uncontrollable bleeding and hemodynamic instability [8].

In our case, the patient has referred to Department of Interventional Radiology for transcatheter arterial embolization due to the hemodynamic stability. Five months after embolization showed significant tumor regression and the disappearance of perinephric hematoma on US scan.

### Conclusion

MDCT is most reliable method on diagnosis of AMLs and for the evaluation of its complications. Transcatheter arterial embolization prefer safely performed while preserving most renal function

### Competing interests

The authors declare that they have no competing interests.

- 1.Lim CH, Mulvin D. Embolisation of bleeding renal angiomyolipoma in pregnancy. Open J Urol 2011;1(3):25-7.
- 2. Ferianec V, Gábor M, Caňo M, Papcun P, Holomáň K. Severe retroperitoneal haemorrhage in the first trimester of a multiple pregnancy after spontaneous rupture of renal angiomyolipoma. Arch Gynecol Obstet 2013;288(6):1193-4.
- 3.Hiromura T, Nishioka T, Tomita K. Spontaneous rupture of renal angiomyolipoma: value of multidetector CT angiography for interventional therapy. Emerg Radiol 2005;12(1-2):53-4.
- 4. Yamakado K, Tanaka N, Nakagawa T, Kobayashi S, Yanagawa M, Takeda K. Renal angiomyolipoma: relationships between tumor size, aneurysm formation, and rupture. Radiology 2002;225(1):78-82.
- 5. Illescas Molina T, Montalvo Montes J, Contreras Cecilia E, Muñoz Muñiz M, González González A, Herraiz Martínez M. Angiomiolipomas, esclerosis tuberosa y gestación: caso clínico. Ginecol Obstet Mex 2009;77(8):380-6.
- 6. Lu CY, Min PQ, Wu B. CT evaluation of spontaneously ruptured renal angiomyolipomas with massive hemorrhage spreading into multi-retroperitoneal fascia and fascial spaces. Acta Radiol Short Rep 2012;1(4). pii: arsr.2012.110009, DOI: 10.1258/arsr.2012.110009.
- 7. Lewis EL, Palmer JM. Renal angiomyolipoma and massive retroperitoneal hemorrhage during pregnancy. West J Med 1985;143(5):675-6.
- 8. Kellner DS, Ercolani MC, Isom-Batz G, Javit DJ, Armenakas NA. Renal angiomyolipoma presenting with massive retroperitoneal hemorrhage. Hosp Physician 2004;40(12):34-6.

#### How to cite this article:

Gundogdu F, Okur A, Serin H.İ, Albayrak S. Importance of Ct Imaging on Spontaneous Rupture of Renal Angiomyolipoma: A Case Report. J Clin Anal Med 2015;6(suppl 5): 656-8.



# **Urinary Retention Associated** with Atomoxetine Use: A Case Report

Atomoksetin Kullanımına Bağlı Üriner Retansiyon / Urinary Retention Associated with Atomoxetine

Nilfer Sahin<sup>1</sup>. Hatice Altun<sup>2</sup>

Çocuk ve Ergen Ruh Sağlığı ve Hastalıkları ABD, Muğla Sıtkı Koçman Üniversitesi Tıp Fakültesi, Muğla <sup>2</sup>Çocuk ve Ergen Ruh Sağlığı ve Hastalıkları ABD, Kahramanmaraş Sütçü İmam Üniversitesi Tıp Fakültesi, Kahramanmaraş, Türkiye

> This paper was presented as a poster at the 7th International Congress on Psychopharmacology @ 3rd International Symposium on Child and Adolescent Psychopharmacology (7th ICP - 3rd ISCAP) held on April, 2015, in Antalya, Turkey.

Atomoksetin en iyi değerlendirilmiş stimülan olmayan tedavi ajanıdır ve geleneksel Dikkat Eksikliği Hiperaktivite Bozukluğu tedavisinde stimülan olmayan ilaçlar arasında etkinliği ve tolere edilebilirliği ile ilk tercih ilaçtır. Ürolojik acillerden biri olan akut idrar retansiyonu, şiddetli idrar yapma hissiyle birlikte mesanenin dolu olmasına rağmen, idrar yapamama halidir. Üriner retansiyonun antikolinerjik etkinliği olan ilaçların kullanımıyla ortaya çıkabileceği bilinmektedir. Bu yazıda atomoksetin tedavisini takip eden süreçte akut üriner retansiyon gelişen 12 yaşında bir erkek olgusu sunulmuştur.

#### Anahtar Kelimeler

Atomoksetin; Yan Etki; Üriner Retansiyon

#### Abstract

Atomoxetine is a well-studied non-stimulant treatment agent, and is also the first-choice non-stimulant drug for the conventional treatment of Attention Deficit Hyperactivity Disorder owing to its effectiveness and tolerability. Urinary retention is a type of urological emergency that is associated with the inability to urinate despite the bladder being full and a strong urge to urinate. It is known that urinary retention can also develop due to the use of anticholinergic drugs. In this manuscript, we present a 12-year-old male case who developed acute urinary retention in the period following atomoxetine treatment.

Atomoxetine; Side Effect; Urinary Retention

DOI: 10.4328/JCAM.3726 Corresponding Author: Nilfer Şahin, Çocuk ve Ergen Ruh Sağlığı ve Hastalıkları ABD, Muğla Sıtkı Koçman Üniversitesi Tıp Fakültesi, 48000, Muğla, Türkiye. T.: +90 2522115177 F.: +90 2522126804 E-Mail: nilfersahin@hotmail.com

Atomoxetine is a well-studied non-stimulant treatment agent, and is also the first-choice non-stimulant drug for the conventional treatment of Attention Deficit Hyperactivity Disorder (ADHD) owing to its effectiveness and tolerability. By inhibiting the presynaptic reuptake of norepinephrine, atomoxetine leads to an increase in the level of norepinephrine. Atomoxetine has a limited effect on the reuptake of serotonin, and minimal affinity to the receptors of other neurotransmitters and carriers. Atomoxetine also affects cognitive functions by increasing the level of dopamine in the prefrontal cortex. Many previous studies have reported that atomoxetine is well tolerated, and that side effects are uncommon [1]. The reported half-life is 5 hours, and it is metabolized mainly by CYP2D6 in the liver. The most common side effects reported among children and adolescents include stomachache, decreased appetite, vomiting, somnolence, nervousness, asthenia, vertigo and dyspepsia [2]. Although it is known that use of atomoxetine at an early age can cause acute urinary difficulties, a review of the literature reveals only a single case in which atomoxetine use led to urinary retention in the child age group [3]. In this manuscript, we present a 12-year-old male case who developed acute urinary retention in the period following atomoxetine treatment.

#### Case Report

A 12-year-old male case was brought by his mother to our outpatient clinic due to the following complaints: "unwillingness to study at home, attention deficit, low performance in class, forgetfulness, hyperactivity, constant and excessive talking, improper behavior in class, impatience." Based on the case's history, the child's problems became even more pronounced after he began school. After being referred to the child psychiatry outpatient clinic by a school counselor while in 2nd class, the child was diagnosed with "ADHD" and started on short-acting methylphenidate 5 mg three times a day. In the following period, the child's treatment was changed to the long-acting form of methylphenidate. However, increased the dose to 36 mg/day led to complaints of excessive palpitations, after which the child's family discontinued the treatment and did not take their children to another psychiatric examination.

The medical history of the case further described that his motor and mental development stages were normal; that he did not experience and epileptic seizure/trauma or other important disease. No specific characteristics or traits were identified in the family history.

During the child's psychiatric evaluation, it was observed that his level of psychomotor activity was more than normally according to his age and developmental level, he had difficulty in focusing his attention, and his level of impulsivity was very pronounced. No further psychiatric signs were identified. Based on the child's psychiatric assessment, a diagnosis of ADHD-Combined Type was considered to patient according to the DSM-IV-TR criteria. As methylphenidate use was associated with complaints of palpitation, the child was referred to a Pediatrician for cardiac evaluation. The physical examination and ECG findings of the patient were within normal limits. And atomoxetine treatment was started. Based on the case's weight (55 kg), it was planned to give atomoxetine at a dose of 25 mg/day, and

to raise the dose to 50 mg/day two weeks later; however, the case experienced a decrease in the frequency and quantity of urination on the first day following the beginning of the treatment, and urinary retention on the second day following the beginning of the treatment. The case reapplied to our outpatient clinic due to this complaint. The case was not taking any medication other than atomoxetine, and had no history of trauma, or any previous complaints of burning sensation during urination or urinary retention. No problem was identified in any of the case's biochemical parameters. The urology department was consulted. The physical examination and ultrasonography performed by the Urology clinic suggested acute urinary retention associated with atomoxetine, and that no relieving discharge was necessary. The case's atomoxetine was discontinued, and a control visit was scheduled with the patient on the following day. During this control visit performed one day later, it was learned that the patient's urination frequency had returned to normal, and that he no longer experienced difficulty in urination.

#### **Discussion**

Urinary retention is a type of urological emergency that is associated with the inability to urinate despite the bladder being full and a strong urge to urinate. Although urinary retention is quite uncommon among children [4]. The two main causes of urinary retention during childhood are neurological diseases and anatomic disorder [5]. It is known that urinary retention can also develop due to the use of anticholinergic drugs [6]. In our case, urinary retention developed immediately after atomoxetine use, and resolved after the atomoxetine treatment was stopped. As all other potential causes of urinary retention were excluded based on the case's medical history and various tests, the patient's urinary retention was determined to be associated with atomoxetine use.

The urinary storage and discharge processes are regulated by the balanced between the sympathetic and the parasympathetic pathways. However, the excessive activation of the sympathetic system may lead to urinary retention through the inhibition of the parasympathetic pathway. Atomoxetine leads to an increase in the level of norepinephrine by inhibiting the presynaptic reuptake of norepinephrine. Urinary retention caused by the use of atomoxetine, a noradrenergic agent, can be explained by the disruption of the sympathetic-parasympathetic balance involved in the process of urination due to the excessive activation of the sympathetic system. It has been reported that serotonin has an inhibitory effect on micturition, and that this effect becomes more pronounced in advancing ages [7]. It is believed that antidepressants which inhibit the reuptake of serotonin can also inhibit micturition by increasing the level of serotonin, and thus lead to urinary retention [8]. As atomoxetine has a certain (yet limited) effect on the reuptake of serotonin, we believe that this effect might have contributed to the urinary retention side effect. Desarkar et al. previously reported an adolescent case who developed urinary retention following the use of 25 mg/g atomoxetine on the 2nd day of treatment, who required relieving discharge, and whose clinical condition resolved following the discontinuation of the drug [3]. Our case similarly developed urinary retention following the administration of low dose atomoxetine; however, the case's urinary retention required no clinical intervention, and resolved by itself following the discontinuation of the drug.

In conclusion; although urinary retention is a rarely observed side effect of atomoxetine, it can still lead to serious consequences. The current case is important in that it illustrates the necessity of clinicians to be cautious about the potential side effects which might develop following atomoxetine administration, as well as the importance of early intervention. However, urinary retention quickly responds to discontinuation of the drug and common clinical intervention for the condition (i.e. catheterization).

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Banaschewski T, Rossner V, Dittmann W. Non stimulant medications in the treatment of ADHD. Eur Child Adolesc Psychiatry 2004;138(suppl.1):102-16.
- 2. Caballero J, Nahata MC. Atomoxetine hydrochloride for the treatment of attention-deficit/hyperactivity disorder. Clin Ther 2003;25(12):3065-83.
- 3. Desarkar P, Sinha VK. Acute urinary retention associated with atomoxetine use. Aust NZJ Psychiatry 2006;40:936.
- 4. Efesoy O, Saylam B, Erdem E. Çocukluk çağında akut idrar retansiyonunun nadir bir nedeni: üretra taşı. Turkiye Klinikleri J Urology 2010;1:60-4.
- 5. Leslie JA, Cain MP. Pediatric urologic emergencies and urgencies. Pediatr Clin North Am 2006;53:513-27.
- 6. Verhamme KM, Sturkenboom MC, Stricker BH, Bosch R. Drug-induced urinary retention: incidence, management and prevention. Drug Saf 2008;31(5):373-88.
- 7. Espey MJ, Downie JW. Serotonergic modulation of cat bladder function before and after spinal transection. European Journal of Pharmacology 1995;287(2):173-
- 8. Lowenstein L, Mueller ER, Sharma S, FitzGerald MP. Urinary hesitancy and retention during treatment with sertraline. Int Urogynecol J Pelvic Floor Dysfunct 2007;18(7):827-9.

### How to cite this article:

Şahin N, Altun H. Urinary Retention Associated with Atomoxetine Use: A Case Report. J Clin Anal Med 2015;6(suppl 5): 659-61.



# Pulmonary Embolism Mimicking Community Acquired Pneumonia: A Case Report

# Toplum Kökenli Pnömoniyi Taklit Eden Pulmoner Emboli Olgusu

Pnömoniyi Taklit EdenPulmoner Emboli / Pulmonary Mimicking Pneumonia

İbrahim Koç¹, Yusuf Doğan², Onur Çakmak³, Gökhan Bulut⁴¹Pulmonary Medicine, ²Microbiology, ³Internal Medicine, ⁴Radiology, Viranşehir State Hospital, Sanlıurfa, Turkey

#### Özet

Akut pulmoner emboli venöz tromboembolinin sık görülen bir formu olup bazı olgularda fatal seyredebilmektedir. Hastalar sıklıkla özgün olmayan şikayet ve klinik bulgularla başvurmakta dolayısıyla tanı koymak zor olabilmektedir. Burada otuz yaşında bir erkek hastada ilk etapta toplum kökenli pnömoni saptanan ancak ileri incelemede pulmoner emboli olduğu anlaşılan bir olguyu sunmayı amaçladık. Otuz yaşında erkek hasta kliniğimize plörötik tarzda sağ yan ağrısı ve nefes darlığı şikayeti ile başvurdu. Akciğer grafisinde sağ akciğerde konsolidasyon ve plevral efüzyon saptandı. Fizik muayenede ateşi 38°C, oda havasında oksijen saturasyonu % 85 olan hastada uygulanan antibiyoterapi ve oksijen tedavisine cevap alınamadı. Akciğerin bilgisayarlı tomografi anjiografisinde sağ akciğerde pnömoni ve pulmoner emboli ile uyumlu bulgular saptandı. Kan ve balgam kültürlerinde üreme olmadı. Kardiyo-Vasküler Hastalıklara Yatkınlık (CVD) panelinde protrombin G20210A ve metilentetrahidrofolat redüktaz (MTHFR) C677T'de heterozigot mutasyon saptandı. Sonuç olarak pulmoner emboli toplum kökenli pnömoniyi taklit edebilmekte dolayısıyla klinisyenlerin tanı sürecinde dikkatli olması gerekmektedir.

#### Anahtar Kelimeler

Bilgisayarlı Tomografi Anjiografi; Pnömoni; Pulmoner Emboli

#### Abstract

Acute pulmonary trombo embolism is a form of venous thromboembolism that is common and sometimes even may be fatal. Patients might present with variable clinical presentation and often have non-specific complaints which make the diagnosis challenging. Here we aimed to report a thirty years old male who was diagnosed with community acquired pneumonia but further investigations revealed pulmonary embolism. A thirty years old male presented to our clinic with right sided chest pain and shortness of breath. Chest radiograph revealed right sided consolidations and pleural effusion. His physical examination revealed high body temperature (38 C°) and oxygen saturation on room air was 85 %. The patient did not respond to the antibiotherapy and oxygen supply. Computed tomography angiography of the chest revealed right sided pulmonary embolism with pneumonia. Blood and sputum cultures revealed no bacteria. Cardiovascular disease panel revealed heterozygous mutation in prothrombine G20210A and metilentetrahidrofolate reductase (MTHFR) C677T. In conclusion pulmonary embolism may mimic community acquired pneumonia thus clinicians must be carefull during the diagnostic process.

#### Keywords

Computed Tomography Angiography; Pneumonia; Pulmonary Embolism

DOI: 10.4328/JCAM.3770 Received: 14.07.2015 Accepted: 06.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 662-4 Corresponding Author: İbrahim Koç. Pulmonary Medicine, Viranşehir State Hospital, Sanlıurfa, Turkey. GSM: +905453142502 E-Mail: İbrahimkoc1981@gmail.com

Pulmonary embolus (PE) refers to obstruction of the pulmonary artery or one of its branches by material (eg, thrombus, tumor, air, or fat) that originated elsewhere in the body. Acute pulmonary embolism is a form of venous thromboembolism (VTE) that is common and sometimes even may be fatal. Patients may present with different variable clinical presentations and often have nonspecific complaints which make the diagnosis challenging. The reported annual incidence of VTE differs ranging between 23-69 cases per 100,000 population, [1, 2] with approximately one third of patients presenting with acute PE and two thirds with deep vein thrombosis [3]. The evaluation of patients with suspected PE should be efficient so that patients can be diagnosed and therapy administered quickly to reduce the associated morbidity and mortality. Community acquired pneumonia may be complicated by pulmonary thromboembolism, but prevalence is low. The basic difficulty is sometimes to differ whether there is pneumonia or it is PE. Here we report a 30 years old male who was admitted to our clinic and had pulmonary embolism mimicking community acquired pneumonia.

### Case Report

A thirty years old male was admitted to our hospital with complaints of right sided chest pain and shortness of breath. His medical and family histories were unremarkable. His physical examination revealed high body temperature (380 C), heart rate of 110 beats per minute, respiratory rate of 20 breaths per minute, blood pressure of 110/80 mm Hg, and SpO2 of 85% on room air. Physical examination of the chest revealed diminished movement of the right hemithorax and dullness over the right lung base along with diminished lung sounds in the right lover zone. His initial blood analyses were as follows: White blood cells;13040/uL (78.7% granulocytes, 8.1% lymphocytes, 12.5% monocytes, 0.7% eosinophils); Hb 12 gm/dL; Hct 36 %; platelets, 580,000/uL, ESR 50 mm/1st hour and serum CRP 200mg/L. Chest X-ray at admission was compatible with right sided pneumonia and pleural effusion. The patient was hospitalised and antibiotherapy started. About 20 mL of serofibrinous pleural fluid was obtained by thoracentesis from the right side under local anesthesia. Biochemical analysis of the pleural fluid revealed an exudative pleural effusion with LDH 320 U/L (normal range: 240-480 U/L), glucose 98 mg/dL, total protein 5.3g/ dL and albumin 2.9 g/dL. Pleural fluid pH was 7.4. Low oxygen saturation, shortness of breath, previously no medical history and sudden onset of the disease aroused suspicion of acute PTE. Computed tomography angiography (CTA) of the chest revealed right sided pulmonary embolism, infiltration and pleural effusion (Figure 1-2). Blood, sputum and pleural fluid cultures revealed no bacteria. Hyperacute onset, no isolation with cultures else more no clinical, radiological and laborotory findings improvement after antibiotherapy was suggestive of pulmonary embolism other than pneumonia. Genetical analysis must be performed when PTE is seen before the age of 40 especially in those who has no previous medical or surgical history and immobilisation. CVD panel of our patient revealed heterozygous mutation in prothrombine G20210A and metilentetrahidrofolate reductase (MTHFR) C677T.



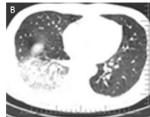


Figure 1. Computed tomography angiography of the chest revealed right sided pleural effusion (A) and pneumonic consolidation (B).

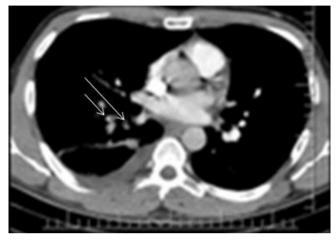


Figure 2. Computed tomography angiography of the chest revealed pulmonary tromboembolism in the right middle pulmonary artery (arrows).

#### Discussion

PTE is a disease that can frequently cause dyspnea, chest pain, fainting, and hemoptysis which can be mimicked by other pulmonary and cardiac diseases. Radiologic features of PTE on plain film are Fleishner sign: enlarged pulmonary artery (20%), Hampton hump: peripheral wedge of airspace opacity and implies lung infarction (20%), Westermark's sign: regional oligaemia (10%), pleural effusion (35%)[4]. On computed tomography angiography; webs or bands, intimal irregularities, abrupt narrowing or complete obstruction of the pulmonary arteries, "pouching defects" which are defined as chronic thromboemboli organised in a concave shape that "points" toward the vessel lumen [4]. It has been reported that prevelance of PTE in patients with pneumonia as high as 10% [5]. It has been shown that recent respiratory infection, acute infection and raised inflammatory markers, are associated with increased risk of thromboembolic disease [6]. Another study has proven that there are strong associations between recent respiratory infection and VTE which may be related to the severity of the infection [6, 7]. Community acquired pneumonia can be complicated by pulmonary thromboembolism, but prevalence is low. Eventhough they may co-exist also may be confused with each other. Our case was a young officer who had no previous medical history and risk factors for PTE. Low oxygen saturation, shortness of breath, previously no medical history and sudden onset of the disease aroused suspicion of acute PTE. Pulmonary embolism may mimic pneumonia, especially in those patients who has no a previos medical history and with acute onset of symptoms. On the other hand there are reported cases with co-existance of both diseases [8]. Clinicians must be carefull, acute onset of symptoms like shortness of breath, pleuritic chest pain, and hypoxemia should arouse suspician.

In conclusion pulmonary embolism may mimic community ac-

quired pneumonia thus clinicians must be careful during the diagnostic process.

#### Competing interests

The authors declare that they have no competing interests.

#### Refereances

- 1. Silverstein MD, Heit JA, Mohr DN, Petterson TM, O'Fallon WM, Melton LJ, 3rd. Trends in the incidence of deep vein thrombosis and pulmonary embolism: a 25year population-based study. Arch Intern Med 1998;158:585-93.
- 2. Anderson FA Jr, Wheeler HB, Goldberg RJ, Hosmer DW, Patwardhan NA, Jovanovic B, et al. A population-based perspective of the hospital incidence and casefatality rates of deep vein thrombosis and pulmonary embolism. The Worcester DVT Study. Arch Intern Med 1991;151:933-8.
- 3. White RH. The epidemiology of venous thromboembolism. Circulation 2003;107(23 Suppl 1):14-8.
- 4. Han D, Lee KS, Franquet T, Muller NL, Kim TS, Kim H, et al. Thrombotic and nonthrombotic pulmonary arterial embolism: spectrum of imaging findings. Radiographics 2003;23:1521-39.
- 5. Rohacek M, Szucs-Farkas Z, Pfortmuller CA, Zimmermann H, Exadaktylos A. Acute cardiac disorder or pneumonia and concomitant presence of pulmonary embolism. PLoS One 2012; DOI: 10.1371/journal.pone.0047418.
- 6. Clayton TC, Gaskin M, Meade TW. Recent respiratory infection and risk of venous thromboembolism: case-control study through a general practice database. Int J Epidemiol 2011;40:819-27.
- 7. Schmidt M, Horvath-Puho E, Thomsen RW, Smeeth L, Sorensen HT. Acute infections and venous thromboembolism. J Intern Med 2011;271:608-18.
- 8. Rokusz L. Case of Legionella pneumonia complicated with pulmonary thromboembolism. Acta Microbiol Immunol Hung 2010;57:401-6.

#### How to cite this article:

Koç İ, Doğan Y, Çakmak O, Bulut G. Pulmonary Embolism Mimicking Community Acquired Pneumonia: A Case Report. J Clin Anal Med 2015;6(suppl 5): 662-4.



## **Transudative Chylothorax** in a Patient with Pulmonary Hypertension



Bu olgu Türk Toraks Derneği'nin 1-5 Nisan 2015 tarihleri arasında yapılan 18. Yıllık kongresinde poster olarak sunulmuştur.

Plevral boşlukta şilöz sıvının toplanması olarak tanımlanan şilotoraks, aralarında pulmoner hipertansiyonun (PH) da bulunduğu çeşitli patolojiler nedeniyle gelişen ve nadir görülen bir klinik tablodur. Şimdiye dek pulmoner hipertansiyona bağlı olarak gelişen sadece bir kaç transüda şilotoraks olgusu bildirilmiştir. Bu sunumda kapak patolojisi ve sağ kalp yetmezliğine bağlı PH sonucu gelişmiş olan, 70 yaşında bir kadında saptanan bir transudatif şilotoraks olgusu sunulmuştur.

#### Anahtar Kelimeler

Transüda Şilotoraks; Pulmoner Hipertansiyon; Sağ Kalp Yetmezliği

Chylothorax, presence of chyle in the pleural space, is an infrequent clinical form of pleural effusion developed due to several pathologies, including pulmonary hypertension. Since now, very few clinical cases of transudative chylothorax due to pulmonary hypertension have been reported. In this report, we present a transudative chylothorax case of 70-year-old female patient with pulmonary hypertension due to cardiac valvular insufficiency and right heart failure.

Transudative Chylothorax; Pulmonary Hypertension; Right Heart Failure

DOI: 10.4328/JCAM.3690 Corresponding Author: Sedat Kuleci, Göğüs Hastalıkları Anabilim Dalı, Çukurova Üniversitesi Tıp Fakültesi, Balcalı, Adana, Türkiye. GSM: +905072516981 E-Mail: skuleci@gmail.com / skuleci@cu.edu.tr

Chylothorax is defined by the presence of chyle in the pleural space. [1, 2]. The accumulation of chyle in the pleural cavity may be due to rupture of the thoracic duct and/or its tributaries, leakage from the pleural lymphatics and/or collateral vessels, or transdiaphragmatic flow of chyle from the peritoneal cavity in patients with chylous ascites [2]. Situations evoking any of these mechanisms represent chylothorax causes (Table 1), which are grouped into four major categories: trauma, malignancy, miscellaneous and idiopathic [1 - 4]. Transudative chylothorax has been reported between %15 - %32 of all chylothorax cases, principally among cirrhosis cases [5, 6]. In this report, we present a case of transudative chylothorax possibly developed from pulmonary hypertension due to cardiac valvular insufficiency and right heart failure.

Table 1. Etiopathogenetic classification of chylothorax

Table 1. Etiopathogenetic classification of Chylotholax				
A. Traumatic	C. Miscellaneous			
a. latrogenic	a. Increased lymph volume +/or			
i. Surgical	abnormal lymphatic lumen			
ii. Non-surgical	i. Lymphatic disorders - congenital			
b. Non-latrogenic	ii. Lymphatic disorders - acquired			
B. Malignancy	b. Lymphatic obstruction			
a. Infiltration	i. Intraluminal obstruction			
b. lymphatic obstruction	ii. External pressure			
	c. Increased venous pressure			
	d. Chylous ascites			
	D. Idiopathic			

#### **Case Report**

A seventy years old, non-smoker female patient admitted to our hospital with symptoms of dyspnea, right-sided chest pain for last 6 months. She had a history of mitral valve replacement in 1993 and she has been anticoagulated since then. Physical examination revealed signs of right-sided pleural effusion. On chest radiography, pleural effusion covering nearly half of the right hemithorax was observed (Figure 1). Hematologic and biochemical laboratory findings were all normal. But, only, the brain

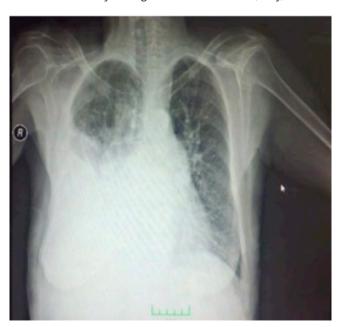


Figure 1. Chest x-ray of the patient at first admission

natriuretic peptide (BNP) level was found very high (4630 pg/mL (N: 0-197 pg/mL)). Echocardiographic evaluation was reported as: "Normal ventricular contractility (Ejection fraction: 60%), prosthetic mitral valve, severe pulmonary hypertension (sPAP: 115mmHg) and tricuspit insufficiency, moderate aortic insufficiency, dilatation of left atrium, and right atrium and ventricle". When first thoracentesis was performed, pleural effusion sample revealed transudative characteristics (Table 2). Microbi-

Table 2. Laboratory findings of two thoracenteses

Charactereristic	1st Thoracentesis			2nd Thoracentesis		
	Pl. Eff.	Serum	Ratio	Pl. Eff.	Serum	Ratio
LDH* [U/L)	44	172	0,25	185	320	0,57
Total Protein [g/dl)	1,7	6,5	0,26	2,7	5,8	0,46
Glucose [mg/dl)	102	115		146	155	
pН	8,0			8,5		
Cholesterol [mg/dl)				41		
Triglyceride [mg/dl)				627		

ologic and cytologic laboratory findings of pleural effusion were found all normal. Calcium channel blockers and diuretics were administered for cardiac problems. In clinical follow-up, on 12th of hospital day, pleural effusion level was found to be increased on chest x-ray and clinically, she was deteriorated (Figure 2).



Figure 2. Chest x-ray on 12th day of admission

Closed chest tube drainage was performed on right hemithorax for relieving excessive pleural effusion. Since the color of pleural effusion in chest tube appeared to be milky, pleural effusion was evaluated biochemically for chylothorax, also. Triglyceride level was found three fold higher than normal, but it was still transudative (Table 2).

Oral feeding is discontinued; a diet with low triglyceride levels was initiated. Somatostatin was ordered at dose of 6 mg/day. She was evaluated for possible malignancies with thoracic-abdominal and pelvic computerized tomography. Radiological and clinical findings were normal except a 5 cm of pleural effusion and enlarged pulmonary artery. The patient was followed for two months in the clinic ward.

After clinical stabilization and radiological improvement are achieved, chest tube drainage discontinued. She was discharged from hospital after two months of treatment period. She has been still following up by out-patient clinics of chest disease.

#### Discussion

Chylothorax is not a common form of pleural effusion. Generally, chylothorax is taken in account, if pleural fluid is in milky appearance. It should be noted that not all chylous pleural effusions appear milky white. Almost 50% of chylothorax cases present as bloody, yellow or green, turbid, serous or serosanguineous effusions [1 - 3]. Chylothoraces are usually characterized by all three of the following: 1.) a triglyceride level of more than 110 mg/dL; 2.) a ratio of pleural fluid to the serum triglyceride level of more than 1.0; and 3.) a ratio of the pleural fluid to serum cholesterol level of less than 1.0 [7].

And also, chylothorax does not always meet the exudative criteria. Transudative form has been reported between %15 - %32 in several studies due to various pathologies [5, 6]. In a review of 2005, 15 cases have been reported as transudative chylothorax, which can be attributed to cirrhosis, nephrosis or heart failure [8]. However, only two cases of transudative chylothorax due to pulmonary hypertension secondary to right heart failure have been presented at "International PHA Conference and Scientific Sessions" in 2014 and "American Thoracic Society International Conference" in 2010. Our patient is a case of transudative chylothorax possibly due to pulmonary hypertension secondary to cardiac valvular insufficiency and right heart failure.

The fundamental mechanism behind chylothorax is the leakage of chyle into the pleural space. Trauma to the thoracic duct is the most common mechanism of chylothorax. Among the patients with chylothorax with a known cause, lymphoma has been considered the most frequent one, followed by metastatic carcinoma [7]. However, the increasing number of intrathoracic surgical procedures and the frequent use of the great veins for total parenteral nutrition and hemodynamic monitoring have contributed to a recent increase in the number of iatrogenic chylothoraces, which may have exceeded the number of those caused by malignancy [3, 4].

The possible mechanism for transudative chylothorax in PAH patients is poorly understood. It is postulated that acute increased right-sided heart pressures led to elevated pressure in the superior vena cava and back pressure into the thoracic duct. Reducing the pressure in the system resolved the chylothorax [8].

Our patient had no recent history trauma or invasive procedure, except thoracentesis and closed tube drainage after hospitalization. Biochemical evaluation of pleural effusion for the second thoracentesis revealed transudative chylothorax (Table 2). All cytological and microbiological evaluations were found normal. On computerized tomography of the whole body; no lymphadenopathy was determined, and neither granulomatous and cystic diseases, nor parenchymal diseases have been detected in thoracic evaluation. On echocardiographic evaluation, right heart failure due to mitral valve disease and pulmonary arterial hypertension (sPAP=115 mmHg) have been detected. With all these clinical, radiological and laboratory findings, patient has been accepted as transudative chylothorax due to PAH.

Therapeutic modalities for chylothorax include oral intake discontinuation, dietary regimens include a high protein-low fat diet with limited oral intake, supplemented with medium chain triglycerides, or fasting plus total parenteral nutrition, somatostatin infusion, thoracic duct embolization, tube drainage, direct ligation of the thoracic duct, mass ligation of the supradiaphragmatic thoracic duct, pleurovenous or pleuroperitoneal shunting, pleurectomy, pleurodesis with glue or talc and radiotherapy [3, 4].

In our patient, we preferred chest tube drainage, oral intake avoidance, somatostatin infusion, diuretics and supplementary modalities. By these treatment options, she was recovered considerably in a period of two months and is still under serial clinical control.

In conclusion, although very rare, evaluation of transudative pleural effusion for chylothorax should be considered in any patient with pulmonary hypertension with pleural effusion in clinical practice.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Huggins JT. Chylothorax and Cholesterol Pleural Effusion. Semin Respir Crit Care Med 2010;31(6):743-50.
- 2. Skouras V, Kalomenidis I. Chylothorax: diagnostic approach. Curr Opin Pulm Med 2010:16:387-93
- 3. McGrath EE, Blades Z, Anderson PB. Chylothorax: Aetiology, diagnosis and therapeutic options. Respir Med 2010:104:1-8.
- 4. Nair SK, Petko M, Hayward MP. Aetiology and management of chylothorax in adults. Eur J Cardiothorac Surg 2007;32:362-69.
- 5. Maldonado F, Hawkins FJ, Daniels CE, Doerr CH, Decker PA, Ryu JH. Pleural fluid characteristics of chylothorax. Mayo Clin Proc 2009;84:129-33
- 6. Agrawal V, Doelken P, Sahn SA. Pleural fluid analysis in chylous pleural effusion. Chest 2008:133:1436-41.
- 7. Romero S. Nontraumatic chylothorax. Curr Opin Pulm Med 2000;6:287-91.
- 8. Diaz-Guzman E, Culver DA, Stoller JK. Transudative chylothorax: report of two cases and review of the literature. Lung 2005;183:169-75.

#### How to cite this article:

Kuleci S, Baydar O, Guzeli E, Hanta İ. Transudative Chylothorax in a Patient with Pulmonary Hypertension. J Clin Anal Med 2015;6(suppl 5): 665-7.



## Methylenetetrahydrofolate Reductase Polymorphisms at Familial Bladder Cancer: Case Report

## Ailesel Mesane Kanserlerinde Metilentetrahidrofolat Redüktaz Polimorfizmleri: Olgu Sunumu

MTHFR Polimorfizmi ve Mesane Kanseri / MTHFR Polymorphism and Bladder Cancer

Gülay Güleç Ceylan<sup>1</sup>, Cavit Ceylan<sup>2</sup>, Seda Yamak<sup>3</sup>

<sup>1</sup>Tıbbi Genetik ABD, Yıldırım Beyazıt Üniversitesi, Tıp Fakültesi,

<sup>2</sup>Üroloji Kliniği, Türkiye Yüksek İhtisas Eğitim ve Araştırma Hastanesi,

<sup>3</sup>Patoloji Kliniği, Türkiye Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Ankara, Türkiye

#### Uzet

Mesane kanseri tüm dünyada erkeklerde en sık görülen 7. kanser türüdür. Türkiye'de ise akciğer kanserinden sonra ikinci, ürogenital tümörler arasında ise birinci sırada görülmektedir. Birçok moleküler epidemiyolojik çalışmada özellikle MTHFR C677T ve A1298C polimorfizmleri ile mesane kanseri arasında ilişki saptanmıştır. Bu vaka sunumunda, transisyonel hücreli mesane kanserine sahip iki kardeşte MTHFR A1298C heterozigositesi saptanmıştır, bu da MTHFR varyantları ve mesane kanseri arasındaki ilişkiyi doğrulamaktadır. Bu bulgu, farklı etnik grupları içeren daha büyük toplulukları içeren prospektif çalışmalarla desteklenebilir.

#### Anahtar Kelimeler

Ailesel Mesane Kanseri; Polimorfizm; Metilentetrahidrofolat Redüktaz Geni

#### Abstract

Bladder cancer is the seventh most common cancer in men in the world, it is the second most seen cancer after lung cancer and the first in urogenital tumours in Turkey. Many molecular epidemiologic studies have been reported to investigate the associations between the MTHFR C677T and A1298C polymorphisms and bladder cancer risk. In this report, a family with transitional bladder cancer have also MTHFR A1298C heterozygosity which supports the association between MTHFR variants and bladder cancer. This finding should be further validated by prospective and larger studies with more diverse ethnic groups.

#### Keywords

Familial Bladder Cancer; Polymorphism; Methylenetetrahydrofolate Reductase Gene

DOI: 10.4328/JCAM.3753 Received: 09.07.2015 Accepted: 13.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 668-70 Corresponding Author: Gülay Güleç Ceylan, Tibbi Genetik ABD, Yıldırım Beyazıt Üniversitesi, Tıp Fakültesi, Atatürk EAH, 06510, Bilkent, Ankara, Türkiye. GSM: +905323778718 F.: +90 3122912728 E-Mail: gulayceylan23@gmail.com

Bladder cancer is the seventh most common cancer in men in the world and the seventeenth in women. According to KIDEM (Cancer Follow-up and Control Center) studies, it is the second most seen cancer after lung cancer and the first in urogenital tumours in Turkey [1]. Bladder cancer usually occurs in 6th and 7th decades. Although the rate of men/women is 3/1, the deaths because of bladder cancer is mostly seen in women (31%) [1]. 90-95% of bladder cancers are transitional cell cancer. Transitional cell bladder cancers are restricted to mucosa and submucosa at the time of diagnosis approximately in 80% of the patients. This is called as non-muscle invasive bladder cancer. 20% of bladder cancer are local forward stage at the time of diagnosis, 20% of them are metastatic. Cigarette smoking is the most important risk factor for bladder cancer, accounting for 50% of cases in men and 35% in women, but the exact mechanism is not yet understood. Cigarette smoke contains some of xenobiotics, including oxidants and free radicals and cigarette smoke exposure was associated with decreased levels of serum and red blood cell folate and vitamin B12 antioxidants [2]. On the other hand, it has been also reported that plasma total homocysteine concentration is higher in smokers than in nonsmokers. According to these findings, the combined effects of smoking with decreased levels of folate and vitamin B12 and an increased level of homocysteine can induce increased chromosomal damage. If it is so, DNA damage induced by smoking may be modulated by the folate metabolic pathway. Folate and methionine metabolism play important roles in DNA synthesis and DNA methylation, their metabolic pathways may affect disease susceptibility. Methylenetetrahydrofolate reductase (MTHFR) and methionine synthase (MS) are two main enzymes involved in the folate metabolism [2]. MTHFR catalyzes the conversion of 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate, this is the predominant form of folate in plasma and provides the methyl group for de novo methionine synthesis through homocysteine remethylation. The C677T and A1298C are two common polymorphisms in the MTHFR gene affecting enzyme activity. Many molecular epidemiologic studies have been reported to investigate the associations between the MTHFR C677T and A1298C polymorphisms and bladder cancer risk [3]. In this case report, we defined a family with bladder cancer who had MTHFR C677T and A1298C polymorphisms. This study can also be a research for folate metabolic genetic variations on risk of bladder cancer development.

## Case Report

A 39-year-old male was admitted to urology clinic because of the complaints of painless clotted hematuria. After the urological examination, he hospitalized for hematuria etiology. A papillary tumor with a diameter of 3cm at the right lateral wall of the bladder was determined in the diagnostic cystoscopy. Tumor was excised. The pathological diagnosis was non-muscle invasive bladder cancer. The patient was followed by control cystoscopies. After 9 months, tumor was detected again in the bladder at the control cystoscopy and it was taken out. The patient's TCC was classified as muscle invasive (Figure 1). Cystectomy and ileal loop operation was performed for the patient. The patient was recovered and discharged in postoperative

10th day. The family of the patient had a bladder cancer story, so he and his family sent to medical genetics department for genetic analysis (Figure 2). Informed consent form of the patients were obtained. We genotyped the patient and his family (pedigree: 9,14,32-proband-,42) for MTHFR 677 and 1298. For genetic analysis, 5 ml of blood was drawn into tubes containing EDTA from each patient. DNA were extracted using a commercial kit (QIAamp DNA mini kit; Qiagen, Hilden, Germany). Genotyping of MTHFR alleles was performed in all subjects by real-time polymerase chain reaction (RT-PCR) using allelic discrimination. The three genotypes were defined as follows: CC, normal homozygous (wild type); CT, heterozygous; and TT mutant homozygous for C677T and AA, normal homozygous (wild type); AC, heterozygous; and CC mutant homozygous for A1298C.

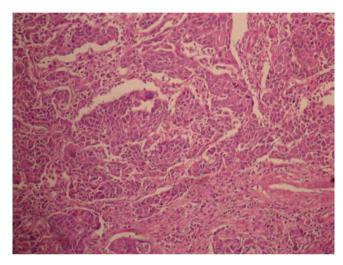


Figure 1. High grade invasive urothelial cancer consists of epithelial islet cells with large hyperchromatin nucleolus which have pleomorphism.

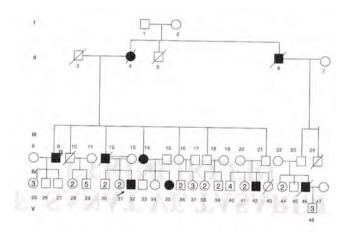


Figure 2. The pedigree of the family.

#### **Discussion**

There is a strong evidence about the interaction between gene variations involved in folate metabolism and risk of bladder cancer. These variations act additively to increase the risk for bladder cancer and this risk is increased among smokers carrying altered genotypes. Many studies focusing on MTHFR variants relationship with the risk of bladder cancer have produced conflicting results. These conflicting results may be explained by the metabolic role of the MTHFR enzyme, which is involved in both DNA methylation and DNA synthesis. The increased risk for the variant MTHFR activity could influence the availability of methyl donors by altering S-adenosyl-methionine levels, and potentially, the methylation status of key tumor suppressor or promoter genes involved in bladder carcinogenesis [3]. Here we report a family with bladder cancer. MTHFR 677\*T and 1298\*C variants in the patients in this family had been performed. The MTHFR 1298 CT genotype was heterozygote in all of the patients in this family who were also diagnosed as transitional bladder cancer. MTHFR 677\*T and 1298\*C variants were both heterozygote in the proband.

In previous studies, for the association between the MTHFR C677T and A1298C polymorphisms and bladder cancer risk, it has been observed that the variant genotype MTHFR 677TT was associated with an increased risk of bladder cancer, compared with the wild-type homozygote 677CC. Folate deficiency leads to decreased DNA methylation and such insufficiency may result in carcinogenesis by inducing genomic instability or activation of oncogenes [2]. In a meta-analysis, 13 different articles were identified to evaluate the association between C677T or A1298C polymorphisms in the MTHFR gene and the risk for bladder cancer. According to this meta-analysis, there was no significant association between the C677T polymorphism and the susceptibility to bladder cancer risk in the overall analysis, but significant relationships were detected in the mixed and Asian populations rather than in Europeans and Africans. This is important, because the allele and genotype distribution of MTHFR C677T locus is different in different races [4]. Safarinejad et al [5] found that the 1298C allele (CA+CC, heterozygotes and homozygotes) was significantly associated with increased risk of bladder cancer in Asians. Similarly, individuals who carried the 1298 CC genotype (homozygote) had a higher risk for bladder cancer in Asians. Moreover, this increased association was also found in Africans. However, the CC genotype (homozygosity) played a protective role for bladder cancer in Europeans [5]. In a study, association between MTHFR C677T and gastric cancer, leucemia and colorectal cancer were also among the most noteworthy associations. Because of its role in a key pathway, the MTHFR C677T variant may have a true impact on cancer risk [6]. In a study by Ozarda et al [13], frequencies of C and T alleles and also frequencies of TT and CC genotypes were investigated in 402 healthy individuals. The frequency of MTHFR T677T genotype was found as 7.7% and the frequency of MTHFR C677T genotype was found as 40%, in males. In females, these rates were as 9.1% and 42.2%, respectively [7]. MTHFR genes play a central role in folate metabolism, and studies have revealed that the cancer risk associated with MTHFR polymorphisms may be modulated by folate intake. The decreased expression of MTHFR by hypermethylation due to the C677 polymorphism may cause an increased risk of DNA hypomethylation of oncogenes, which may not be corrected by other DNA repair enzymes, resulting in a higher susceptibility to bladder cancer in carriers of the 677TT genotype [8].

In conclusion, according to the literature, the MTHFR C677T and A1298C polymorphisms have an effect on increasing risk of bladder cancer [7]. In this report, a family with transitional bladder cancer have also MTHFR A1298C heterozygosity which supports the association between MTHFR variants and bladder

cancer. This finding should be further validated by prospective and larger studies with more diverse ethnic groups and more detailed environmental exposure data.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Ferlay J, Bray F, Pisani P, Parkin DM. GLOBOCAN 2012: Cancer incidence, mortality and prevalence worldwide. IARC Cancer Base, Lyon; 2012.
- 2. Tungtrongchitr R, Pongpaew P, Soonthornruengyot M, Viroonudomphol D, Vudhivai N, Tungtrongchitr A, et al. Relationship of tobacco smoking with serum vitamin B-12, folic acid and haematological indices in healthy adults. Public Health Nutr 2003;6(7):675-81.
- 3. Rouissi K, Ouerhani S, Oliveira E, Marrakchi R, Cherni L, Ben Othman F, et al. Polymorphisms in one-carbon metabolism pathway genes and risk for bladder cancer in a Tunisian population. Cancer Genet Cytogenet 2009;195(1):43-53.
- 4. You W, Li Z, Jing C, Qian-Wei X, Yu-Ping Z, Weng-Guang L, et al. MTHFR C677T and A1298C Polymorphisms were associated with bladder cancer risk and disease progression: A meta-analysis. DNA Cell Biol 2013;32(5):260-7.
- 5. Safarineiad MR. Shafiei N. Safarineiad S. Genetic susceptibility of methylenetetrahydrofolate reductase (MTHFR) gene C677T, A1298C, and G1793A polymorphisms with risk for bladder transitional cell carcinoma in men. Med Oncol 2011;28(1):398-412
- 6. Dong LM, Potter JD, White E, Ulrich CM, Cardon LR, Peters U. Genetic susceptibility to cancer: the role of polymorphisms in candidate genes. JAMA 2008;299(20):2423-36.
- 7. Ozarda Y,Sucu DK, Hizli B, Aslan D. Rate of T alleles and TT genotype at MTH-FR 677C-T locus or C alleles and CC genotype at MTHFR 1298A-C locus among healthy subjects in Turkey: impact on homocysteine and folic acid status and reference intervals. Cell Biochem Funct 2009:27(8):568-77.
- 8. Karagas MR, Park S, Nelson HH, Andrew AS, Mott L, Schned A, et al. Methylenetetrahydrofolate reductase (MTHFR) variants and bladder cancer: a populationbased case-control study. Int J Hyg Environ Health 2005;208(5):321-7.

#### How to cite this article:

Ceylan G.G, Ceylan C, Yamak S. Methylenetetrahydrofolate Reductase Polymorphisms at Familial Bladder Cancer: Case Report. J Clin Anal Med 2015;6(suppl 5): 668-70.



## Watershed Cerebral Infarction in a Patient with Acute Renal Failure

Böbrek Yetmezliği ile Watershed İnfarkt / Watershed Infarction with Renal Failure

Ruya Ozelsancak<sup>1</sup>, Ertugrul Erken<sup>1</sup>, Semih Giray<sup>2</sup>, Ozlem Alkan<sup>3</sup> Departments of 'Nephrology, 'Neurology, 'Radiology, Baskent University Faculty of Medicine Adana Medical and Research Center, Adana, Türkiye

Akut böbrek yetmezliği duygu durum değişikliği, konsantrasyon bozukluğu, tremor, stupor, koma, asteriksis,dizartri gibi nörolojik bulgulara neden olabilir. Bu bulgular aynı zamanda serebral infarktın da bir belirtisidir. Burada, kontrast ve anjiotensin dönüştürücü enzim inhibitörü kullanımına sekonder gelişen akut böbrek yetmezliği olan 70 yaşındaki kadın hastada görülen watershed serebral infarkt vakasını sunduk. Hasta dizartri nedeniyle magnetik rezonans görüntüleme ile değerlendirildi. Görüntülemede her iki serebral hemisferin frontal ve parietal derin beyaz cevherinde, iç sınır bölgesini etkileyen watershed serebral infarkt ile uyumlu milimetrik akut iskemik lezyonlar saptandı. Yatışının beşinci gününde böbrek fonksiyonları normale döndü (BUN 32 mg/dl, kreatinin 1.36 mg/dl) ve hasta taburcu edildi. Dizartri yirmi gün devam etti.

Akut Böbrek Yetmezliği; Dizartri; Watershed İnfarkt

Acute renal failure can cause neurologic manifestations such as mood swings, impaired concentration, tremor, stupor, coma, asterixis, dysarthria. Those findings can also be a sign of cerebral infarct. Here, we report a case of watershed cerebral infarction in a 70-year-old female patient with acute renal failure secondary to contrast administration and use of angiotensin converting enzyme inhibitor. Patient was evaluated with magnetic resonance imaging because of dysarthria. Magnetic resonance imaging revealed milimmetric acute ischemic lesion in the frontal and parietal deep white matter region of both cerebral hemisphere which clearly demonstrated watershed cerebral infarction affecting internal border zone. Her renal function returned to normal levels on fifth day of admission (BUN 32 mg/dl, creatinine 1.36 mg/dl) and she was discharged. Dysarthria continued for 20 days.

#### Kevwords

Acute Renal Failure; Dysarthria; Watershed Infarction

Corresponding Author: Ruya Ozelsancak, Nefroloji BD, Başkent Üniversitesi Tıp Fakültesi, Adana Uygulama ve Araştırma Hastanesi, Yüreğir, Adana, Türkiye. T.: +90 3223444444 F.: +90 3223444445 GSM: +905325864019 E-Mail: rusancak@hotmail.com / rusancak@hotmail.com

Acute renal failure can cause neurologic manifestations. Some of those symptoms are mood swings, impaired concentration, tremor, stupor, coma, asterixis, dysarthria, myoclonus [1]. Similar findings can be a sign of cerebral infarct. Here, we report a case of watershed (WS) cerebral infarction in a patient with acute renal failure secondary to contrast administration and use of angiotensin converting enzyme inhibitor.

#### Case Report

A 70-year-old female patient was admitted to the emergency department with abdominal pain and gastroenteritis which was continuing for one day. She had a history of hypertension and coronary artery disease and was taking perindopril 4 mg/day and metoprolol 50 mg/day for 3 days. She had a coronary angiography 3 days ago. Her medical history included no neurological or cerebrovascular disease. On her physical examination, blood pressure was 100/60 mmHg, pulse 88/min, axillary temperature 37.3 C there were no ralles, S3 or soufflé, bowel sounds has increased. Her laboratory parameters were as below; glucose 139 mg/dl, BUN 61 mg/dl, creatinine 4.51 mg/dl, Na 131 mEq/L, potassium 5.34 mEq/L, calcium 8.35 mg/dl, phosphorus 6.7 mg/dl, ALT 24 IU/L, AST 33 IU/L, amylase 44 U/L, lipase 20 U/L, total protein 5.76 g/dl, albumin 3.7 g/dl, hemoglobin 9.13 g/dl, platelets 423 K/mm3, leukocyte 18 K/mm3, CRP 121 mg/L, PH 7.34, HCO3 25. Examination of urine showed density 1012, protein 0.9 g/L, 4-6 erythrocytes and 2-4 leukocytes. Analysis of stool showed amebic cysts. She was admitted to the nephrology clinic due to acute renal failure secondary to use of angiotensin converting enzyme inhibitor, contrast nephropathy and gastroenteritis. She was treated with amlodipine 10 mg/ day, metronidazole 1500 mg/day and hydrated by serum physiologic and dextrose. Diuresis was between 2000-2500 ml /day. Abdominal ultrasonography showed biliary sludge and kidneys were in normal range. On the second day after hospitalization she became dysarthric, uremic encephalopathy was not sought because her BUN and creatinine were in the previous range. Cranial magnetic resonance imaging was performed to exclude cerebrovascular accident. Magnetic resonance imaging revealed milimmetric acute ischemic lesion in the frontal and parietal deep white matter region of both cerebral hemisphere and in the left parietal post central gyrus, which clearly demonstrated WS infarction affecting internal border zone Figure 1, Figure 2. Carotid and vertebral doppler ultrasonography showed no occlusion. Acetyl salicylic acid 150 mg/day was started. Her renal function returned to normal levels on fifth day of admission (BUN 32 mg/dl, creatinine 1.36 mg/dl) and she was discharged. Dysarthria approximatelly continued for 20 days.

#### **Discussion**

Watershed infarctions involve the junction of distal fields of two nonanastomosing arterial systems. There are two distinct supratentorial WS areas. The first between the cortical territories of the anterior cerebral artery (ACA), middle cerebral artery (MCA) and posterior cerebral artery and those have been commonly referred to as cortical WS areas. The second, between the deep and the superficial arterial system of the MCA or between the superficial system of the MCA and ACA and those

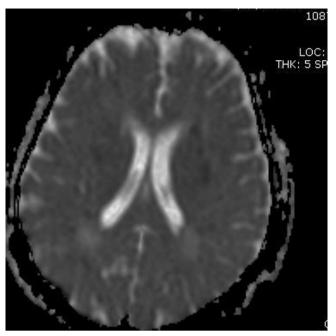


Figure 1. Diffusion-weighted magnetic resonance imaging.

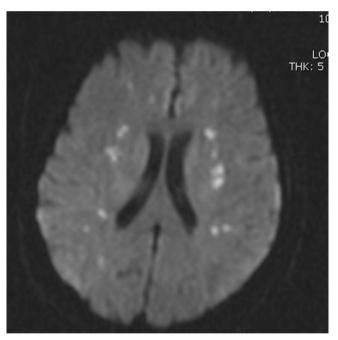


Figure 2. ADC map show multiple acute ischemic lesions in deep white matter region of both cerebral hemispheres

have been referred as the internal WS areas and it take place in the white matter along lateral ventricule [2]. The pathophysiology has not been fully elucidated. A commonly accepted hypothesis is that decreased perfusion in the distal region of the vascular territories leaves them vulnerable to infarction [3]. Severe hypotension can cause bilateral WS infarction. The symptoms of WS infarcts are syncope, episodic, fluctuating or progressive weakness of hand occasionally associated with upper limb shaking. In our case none of those symptoms were observed, dysarthia was the primary symptom. Our patient was using perindopril 5 mg/day for three days and she had a history of gastroenteritis. After all, episodes of hypotension could be the cause of WS infarction. On the other hand we don't know if she had hypotensive or hypertensive episodes during coronary angiography, which makes more vulnerable the WS infarction areas to subsequent events. It has been previously reported a case

of WS infarction in a hemodialysis patient who had recurrent episodes of intradialytic hypotension [4]. Renal failure can also cause neurologic manifestations such as mood swings, impaired concentration, tremor, stupor, coma, asterixis, dysarthria, myoclonus. Advanced uremic encephalopathy is usually characterized by a reduced level of consciousness, anorexia, asterixis, myoclonus, and upper motor neuron signs that result in disturbances of gait and speech. Twenty percent of patients with acute kidney injury in an intensive care unit setting develop neurologic impairment [1]. The treatment of those patients is renal replacement therapy. But in a patient with acute renal failure and stable renal function, as in our case, a careful search for other causes should be initiated before it is considered a clinical feature of uremia requiring renal replacement therapy.

In conclusion, in case of stable renal function, every patient with renal failure and symptoms of uremic encephalopathy shoud be evaluated for cerebral disorders. We could save the patients from unnecessary procedures and complications

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Seifter JL. Neurologic complications of chronic kidney disease. In: Feehaly J. Floege J, Johnson RJ, editors. Comprehensive Clinical Nephrology. 3rd ed. Philadelphia: Mosby Elsevier; 2007.p.887-91
- 2. Momjian-Mayor I, Baron JC. The pathophysiology of watershed infarction in internal carotid artery disease: review of cerebral perfusion studies. Stroke 2005;36(3):567-77
- 3. Mangla R, Kolar B, Almast J, Ekholm SE. Border zone infarcts: pathophysiologic and imaging characteristics. Radiographics 2011;31(5):1201-14.
- 4. Davenport A, Buscombe JR. Watershed cerebral infarction in a hemodialysis patient. Kidney Int 2010;77(12):1140

#### How to cite this article:

Ozelsancak R, Erken E, Giray S, Alkan O. Watershed Cerebral Infarction in a Patient with Acute Renal Failure. J Clin Anal Med 2015;6(suppl 5): 671-3.



# Our Anesthesia Experience for Lower Extremity Surgery in a Patient with Polymyositis

## Polimiyozitli Bir Hastada Alt Ekstremite Cerrahisinde Anestezi Deneyimimiz

Bir Polimiyozit Hastasında Anestezi / Anesthesia in a Polymyositis Patient

Yeliz Kılıç, Tuğba Aşkın, Tülay Dal, Tünay Kandemir, İdil Kaya Department of Anesthesiology and Reanimation, Dr. Abdurrahman Yurtaslan Oncology Training and Research Hospital, Ankara, Turkey

#### Ozet

Polimiyozit ilerleyici kas güçsüzlüğü ile karakterize idiyopatik inflamatuvar miyopatilerin alt tiplerinden biridir. İnterstisyel akciğer hastalığı, toraks kaslarının zayıflığına bağlı pulmoner disfonksiyon, aritmi, dilate kardiyomiyopati ve düşük ejeksiyon fraksiyonu gibi çeşitli pulmoner ve kardiyak bulgular bu hastalığa eşlik edebilir. Polimiyozit hastalarının anestezi yönetiminde, aspirasyon pnömonisi, interstisyel akciğer hastalığı, kardiyak ritim bozuklukları, miyokardit ve kalp yetmezliği gibi eşlik eden sistemik bulguların varlığı ile kas gevşetici ajanların kullanımının potansiyel riskleri başlıca kaygılardır. Bu nedenle, bu hastaların anestezi yönetimi daha fazla dikkat ve yakın monitorizasyon gerektirir. Kendi olgumuzda da olduğu gibi, kas gevşetici kullanımı olmaksızın laringeal maske uygulaması seçilmiş vakalarda güvenle tercih edilebilir.

#### Anahtar Kelimeler

Anestezi; Santral Kas Gevşeticiler; Polimiyozit

#### Abstract

Polymyositis is one of the subtypes of idiopathic inflammatory myopathies characterized by progressive muscle weakness. Several pulmonary and cardiac manifestations such as interstitial lung disease, pulmonary dysfunction due to weakness of thoracic muscles, arrhythmias, dilated cardiomyopathy, and low ejection fraction can be accompanied this disease. The major concerns in the anaesthetic management of the patients with PM are the presence of various systemic disorders such as aspiration pneumonia, interstitial lung disease, cardiac arrhythmia, myocarditis and heart failure, and the potential risks of the use of muscle relaxant agent. Therefore, the anaesthetic management of these patients requires more attention and close monitoring. As in our case, laryngeal mask airway placement without using any muscle relaxant can be safely preferred in selected cases.

#### Keywords

Anesthesia; Central Muscle Relaxants; Polymyositis

DOI: 10.4328/JCAM.3777 Received: 19.07.2015 Accepted: 21.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 674-6 Corresponding Author: Yeliz Kılıç, Department of Anesthesiology and Reanimation, Dr. Abdurrahman Yurtaslan Oncology Training and Research Hospital, 06200, Ankara, Turkey. GSM: +905053573180 F: +90 3123340352 E-Mail: yeliz\_kilic3@hotmail.com

Idiopathic inflammatory myopathies are a heterogeneous group of autoimmune connective tissue disorders characterized by progressive weakness in the proximal striated muscles. It consists of four major subtypes including polymyositis (PM), dermatomyositis, necrotizing myopathy and inclusion body myositis. Among those, PM usually presents with subacute/chronic and symmetric proximal limb weakness, with a prevalence of approximately 10 cases per 100.000 persons in the general population [1]. Several pulmonary, cardiac and systemic manifestations such as interstitial lung disease, pneumomediastinum, pulmonary dysfunction due to weakness of thoracic muscles, arrhythmias, dilated cardiomyopathy, low ejection fraction, fever, and arthralgia can be accompanied PM [2]. The major concerns in the anaesthetic management of the patients with PM are delayed recovery from muscle relaxation, aspiration pneumonia, arrhythmia and cardiac failure [2]. In this paper, we aimed to present our anaesthetic management, particularly the choice of anesthesia method and the use of anaesthetic drugs intraoperatively, in an adult patient who underwent an emergent orthopedic surgery of lower extremity.

#### Case Report

A 38-year-old man, 170 cm in height and 94 kg in weight, presented with open reduction and internal fixation for body fracture of his left tibia. He was diagnosed as PM six years ago, and was treated with prednisolone regularly since then. However, he did not receive any medication for the last six months due to the remission of the disease. On examination, he had mild muscle weakness mainly in the lower limbs, which had been stable for the last year. His cardiac and pulmonary examinations were unremarkable. Echocardiography showed normal cardiac functions with an ejection fraction of 62%. The patient was also consulted by a cardiologist, and no cardiac abnormality was detected. All routine laboratory tests were in normal limits, except elevated creatine kinase (CK 210 U/L, normal ranges: ≤ 145 U/L), aspartate aminotransferase (AST 109 U/L, normal ranges: ≤ 35 U/L) and alanine aminotransferase (ALT 168 U/L, normal ranges: ≤ 40 U/L). Non-specific infiltrations were detected on Chest X-ray. His blood pressure, heart rate, EtCO2, and oxygen saturation were 130/80 mmHg, 94/min, 40 mmHg, and 98% respectively. Airway as-sessment was consistent with Mallampati class 2. Written informed consent form was obtained from the patient for publication of this case report.

In the operating room, monitoring included electrocardiography, noninvasive blood pressure and pulse oximetry. After premedication with midazolam (0.02 mg/kg IV), anesthesia was induced by fentanyl (1  $\mu g/kg$ ), lidocaine (0.5 mg/kg) and propofol (2 mg/kg) with 100% oxygen. Then, a 5 no laryngeal mask was placed to control airway. Anesthesia was maintained with sevoflurane 2% with nitrous oxide (50%) in oxygen (50%) at 6 I/min total gas flow. Before the surgical incision, tenoxicam (20 mg) and ranitidine (50 mg) were given to patient for preemptive analgesia. Prednisolone (1 mg/kg) was also administered. Propofol (0.5 mg/kg) was added two times during the operation. No centrally acting muscle relaxant was used for induction and maintenance anesthesia. The operation lasted 90 minutes without any complication, and finally the laryngeal mask was

removed successfully. The patient was given tramadol (1 mg/ kg) for postoperative pain control. The postoperative course was also uneventfully, and he was discharged on eleventh day.

#### Discussion

The anaesthetic management of the patients with PM poses a concern for anesthesiologists due to several reasons including delayed recovery from muscle relaxation, hyperkalemia, cardiac and pulmonary complications. However, there are only few reports on the issue, which were mostly reported as single cases

It is well known that PM is a systemic disorder presenting with progressive loss of skeletal muscle function. Although neck, shoulder, and pelvic muscle weakness are mostly found in these patients, any striated muscle such as intercostal, diaphragmatic and pharyngeal muscles may also be affected [3]. It is fact that most anaesthetic agents routinely used in general practice have direct or indirect effects on muscles and nerves, therefore it is very important to have sufficient knowledge on the management of the patients with neuromuscular disease such as PM, to avoid potential pitfalls. In this regard, the appropriate use of muscle relaxants is of great importance for the successful anaesthetic management. Although shorter acting neuromuscular blocking drugs such as atracurium may be advised to use for muscle paralysis with slightly increased sensitivity, these patients are believed to be sensitive to nondepolarizing muscle relaxants, and the use of these neuromuscular blockers may cause prolonged muscle weakness and life-threatening cardiac dysrhythmias [6]. While some authors preferred no muscle relaxants for the anaesthetic management of their patients with PM, the others reported titrated small dose of muscle relaxant in conjunction with a close peripheral neuromuscular monitorization as a safe option for such patients where the use of neuromuscular blockers is unavoidable [2,4]. Additionally, the patients with PM generally use steroids as mainstay treatment, and steroid induced myopathy may lead to an increased sensitivity to neuromuscular blocking agents, with unpredictable responses. Immunosuppressants, such as azathioprine and methotrexate, are the second line agents in the treatment of PM, and the interaction between these drugs and non-depolarising muscle relaxants may cause resistance to neuromuscular blocking drugs. Succinylcholine, a depolarising muscle relaxant, is not also recommended because of its potential to malignant hyperthermia and hyperkalemia [7]. In addition, thoracic epidural anesthesia was recommended as a successful anaesthetic method for thoracic surgery [5]. Accordingly, in the present case, we performed laryngeal mask airway placement to avoid the potential risks of general anesthesia, and used no depolarizing or non-depolarizing muscle relaxant agent in both induction and maintenance of anesthesia.

Another important point in the evaluation of polymyositis patients is to determine their cardiopulmonary sufficiencies. Interstitial lung disease is the major pulmonary manifestation, and is usually associated with significant morbidity and mortality. In addition, cardiac involvement may be found in some patients with PM, as arrhythmia, myocarditis or heart failure. Therefore, a detailed preoperative evaluation of cardiopulmonary function should be required to prevent potential severe complications.

In conclusion, the anaesthetic management of patients with neuromuscular disease such as PM requires more clinical attention, particularly in terms of accompanying systemic manifestations and use of muscle relaxant drugs. For our opinion, laryngeal mask airway placement without using any muscle relaxant can be safely preferred in selected cases.

#### Competing Interests

The authors declare that they have no competing interests.

#### References

- 1. Furst DE, Amato AA, lorga SR, Gajria K, Fernandes AW. Epidemiology of adult idiopathic inflammatory myopathies in a U.S. managed care plan. Muscle Nerve 2012;45(5):676-83.
- 2. Ahn S, Lee JH, Yang EA, Park C, Shin DW. Anesthesia for flap surgery in a patient with polymyositis. Korean J Anesthesiol 2014;67(Suppl):S79-80.
- 3. Gunusen I, Karaman S, Nemli S, Firat V. Anesthetic management for cesarean delivery in a pregnant woman with polymyositis: A case report and review of literature. Cases J 2009;2:9107.
- 4. Ohta M, Nishikawa N, Kida H, Miyao S. Anesthetic management of two patients with polymyositis. Masui 2000;49(12):1371-3.
- 5. Yen CR, Tsou MY, Lin SM, Chan KH, Chu YC. Thoracic epidural anesthesia for a polymyositis patient undergoing awake mini-thoracotomy and unroofing of a huge pulmonary bulla. Acta Anaesthesiol Taiwan 2008;46(1):42-5.
- 6. Shikha S, Lakshmi J, Nitin S, Jayashree S: Anaesthetic management for laparoscopic cholecystectomy in two patients with biopsy proven polymyositis. Indian J Anaesth 2007;51(1):43-6.
- 7. Garg R, Bhalotra AR, Bhadoria P, Anand R, Kumar A: Muscle Disorder-Experience with two rare cases. J Anaesth Clin Pharmacol 2008;24(2):225-8.

#### How to cite this article:

Kılıç Y, Aşkın T, Dal T, Kandemir T, Kaya İ. Our Anesthesia Experience for Lower Extremity Surgery in a Patient with Polymyositis. J Clin Anal Med 2015;6(suppl



## Multiple Myocardial Bridges in a Patient with Hypertrophic Cardiomyopathy

Çoklu Miyokardiyal Kas Bandı / Multiple Myocardial Bridges

Tolga Demir<sup>1</sup>, Mazlum Sahin<sup>2</sup>, Semih Öztürk<sup>3</sup>, Hale Bolgi Demir<sup>4</sup>, Ergun Demirsoy<sup>1</sup> Department of Cardiovascular Surgery, Kolan International Hospital, <sup>2</sup>Department of Cardiovascular Surgery, Haseki Training and Research Hospital, <sup>3</sup>Department of Cardiology, Haseki Training and Research Hospital, <sup>4</sup>Department of Cardiovascular Surgery, Medicana International Hospital, İstanbul, Türkiye

Miyokardiyal kas bandı nadir rastlanan konjenital bir anomalidir. Hemen her zaman sol ön inen koroner arterde görülür. Aynı hastada, farklı koroner arterlerde, birden fazla kas bandının eşzamanlı görüldüğü çok az vaka bildirilmiştir. Bu makalede , 60 yaşındaki, hipertrofik kardiyomiyopatili bir erkek hastada, sol ön inen arter ile sağ koroner arterde eşzamanlı olarak görülen miyokardiyal kas bandını sunduk. Miyokard perfüzyon sintigrafisinde iskemi saptanmamış ve hasta medikal olarak takip edilmiştir.

## Anahtar Kelimeler

Miyokardiyal Kas Bandı; Hipertrofik Kardiyomiyopati; Tünel Arter

Myocardial bridging is a rare congenital anomaly. It is almost always confined to the left anterior descending coronary artery. There are few reported cases with simultaneous occurrence of multiple bridges of different coronary arteries in the same patient. In this article we report a 60-year-old male with hypertrophic cardiomyopathy having multiple myocardial bridges left anterior descending and right coronary arteries. No ischemia was detected on myocardial perfusion single photon emission computed tomography and the patient was medically followed up.

Myocardial Bridge; Hypertrophic Cardiomyopathy; Tunnel Artery

DOI: 10.4328/JCAM.3780 Corresponding Author: Tolga Demir, Adana Kahveci Mahallesi, Osmanli Caddesi, No: 17, Gurpinar, Beylikduzu 34528, Istanbul, Turkey. GSM: +905327491319 T.: +90 2126096111 E-Mail: dr.tolgademir@gmail.com

Myocardial bridging is a rare congenital anomaly which epicardial coronary artery is compressed by a muscular band. It is rarely associated with serious complications [1]. Coronary involvement other than left anterior descending artery (LAD) is very rare. Our case presents concomitant bridging of LAD and posterior descending branch of the right coronary artery in a patient with hypertrophic cardiomyopathy.

#### Case Report

60 year old male presented at outpatient clinic complaining of exercise induced dyspnea. He had a history of hypertension and diabetes mellitus. Physical examination did not reveal any remarkable finding. Negative precordial T waves were prominent on electrocardiogram. Transthoracic echocardiography displayed non-obstructive hypertrophic cardiomyopathy (Fig 1). Diastolic septal thickness was measured 36 millimeters.

Given the patient's findings coronary angiogram was performed. Left angiogram showed systolic complete compression of first large septal and mid LAD (Fig 2). Right angiogram showed complete occlusion of distal posterior descending artery during systole as well (Fig 3). During diastole Total recovery and decompression was observed in both coronary arteries. No ischemia was detected on myocardial perfusion SPECT (single photon emission computed tomography). Patient is still being followed up medically in our outpatient clinic on beta blocker and calcium channel blocker therapy.

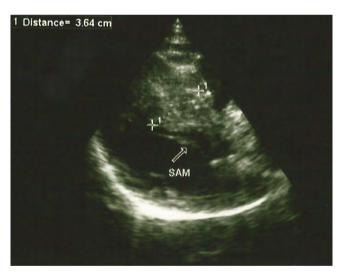


Figure 1. Transthoracic echocardiography showing non-obstructive hypertrophic cardiomyopathy and diastolic septal thickness.

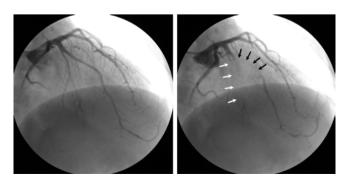


Figure 2. Left angiogram; (A) normal daistolic angiogram, (B) systolic complete occlusion of first septal branch (white arrow) and mid LAD (black arrow).

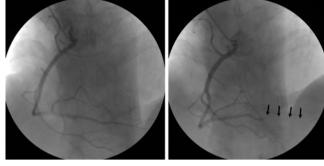


Figure 3. Right angiogram; (A) normal diastolic angiogram, (B) systolic complete occlusion of distal posterior descending artery.

#### Discussion

Myocardial bridging is a congenital variant of a coronary artery in which a portion of an epicardial coronary artery (most frequently the middle segment of the LAD) takes intramural course. This frequently results in vessel compression during systole. Coronary artery coursing within the myocardium is called tunnel artery and the muscle fiber overlying is called myocardial bridge. While frequently asymptomatic, this condition may be responsible for complications including, myocardial ischemia, arrhythmias, left ventricular dysfunction, acute coronary syndrome and even sudden death [1,2].

Since only the symptomatic cases were diagnosed with conventional angiography, prevalence were underestimated. However autopsy series reported higher prevalence up to %80 [1]. Widespread use of computed tomography angiography revealed precise anatomy and also pathophysiology [2]. Despite reported cases of myocardial ischemia ,infarction and sudden death, bridging is mostly accepted a benign entity. New reports raised suspicion of increased atherosclerosis proximal to the bridge [1]. LAD is the most involved segment.

Treatment options remain limited. Medications such as betablockers and calcium channel-blockers remain the first-line therapy. Surgical myotomy reserved for refractor cases. Stenting of the tunneled segment has also been used; however stent failure rate in approximately 50% of the cases, including stent fracture, and coronary perforations have been reported. As with surgical intervention, percutaneous coronary intervention should only be considered as a therapeutic option in patients with bridging refractory to medical therapy, with the expectation that revascularization rates will be high even with drugeluting stents [1].

Depending on the large series including conventional angiography, computed tomography angiography and autopsy data, involvement other than LAD is very rare [2]. The most frequent site of myocardial bridge is found in the middle segment of the LAD. Mavi et al [3] reported that out of 28 patients with myocardial bridge, only 1 had myocardial bridge of the circumflex coronary artery (3.4%). Few case reports presented concomitant RCA and LAD bridging [4,5]. Angelini et al [6] reported that out of 61 patients with myocardial bridge, only 1 had an additional bridge of the posterior descending branch of the right coronary artery.

Hypertrophic cardiomyopathy is a genetic disorder and in these cases, myocardial bridging is the most frequent encountered coronary artery lesion. Although simultaneous occurrence of bridging and hypertrophic cardiomyopathy is frequently report-

ed, and the prevalence of this combination is unclear. Wu et al [7] reported concomitant posterior descending artery and LAD bridging in a patient with hypertrophic cardiomyopathy. Myocardial bridging in adults with hypertrophic cardiomyopathy is often a benign condition, and in one large cohort it was found to have no impact on overall outcome [8]. However, as hypertrophic cardiomyopathy and myocardial bridging are both the leading causes of sudden cardiac death, their combination must alert the clinician. Therefore a general principle, the need for a personalized approach to each individual should be emphasized. This case demonstrates myocardial bridges could rarely involve multiple coronaries and this multi site involvement is associated with hypertrophic cardiomyopathy.

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Loukas M, Von Kriegenbergh K, Gilkes M, Tubbs RS, Walker C, Malaiyandi D et al. Myocardial bridges: A review. Clin Anat 2011;24(6):675-83.
- 2. Ishikawa Y, Kawawa Y, Kohda E, Shimada K, Ishii T. Significance of the anatomical properties of a myocardial bridge in coronary heart disease. Circulation journal : official journal of the Japanese Circulation Society 2011:75(7):1559-66.
- 3. Mavi A, Sercelik A, Ayalp R, Karben Z, Batyraliev T, Gumusburun E. The angiographic aspects of myocardial bridges in Turkish patients who have undergone coronary angiography. Ann Acad Med Singapore 2008;37(1):49-53.
- 4. Kucukosmanoglu M, Akkaya E, Ardic I, Sincer I. Myocardial bridging of the left anterior descending coronary artery and right coronary artery in a patient with mitral valve stenosis. Kardiol Pol 2012;70(6):646-7.
- 5. Hu XY, Zhou DX, Qian JY, Zhang F, Pan CZ, Ge JB. Multiple myocardial bridges affecting left anterior descending artery and right coronary artery with hypertrophic cardiomyopathy: a case report. Chin Med J (Engl) 2007;120(8):734-6.
- 6. Angelini P. Trivellato M. Donis I. Leachman RD. Myocardial bridges: a review. Prog Cardiovasc Dis 1983:26(1):75-88.
- 7. Wu QY, Xu ZH. Surgical treatment of myocardial bridging: report of 31 cases. Chin Med J (Engl) 2007;120(19):1689-93.
- 8. Sorajja P, Ommen SR, Nishimura RA, Gersh BJ, Tajik AJ, Holmes DR. Myocardial bridging in adult patients with hypertrophic cardiomyopathy. Journal of the American College of Cardiology 2003;42(5):889-94.

#### How to cite this article:

Demir T, Şahin M, Öztürk S, Demir H.B, Demirsoy E. Multiple Myocardial Bridges in a Patient with Hypertrophic Cardiomyopathy. J Clin Anal Med 2015;6(suppl 5):



# A Case Report: Osteomyelitis of Calcaneus Caused by Achromobacter Spp.

## Achromobacter Spp.'nin Etken Olduğu Bir Kalkaneus Osteomyeliti

Osteomyelit ve Achromobacter Türleri / Osteomyelitis and Achromobacter Species

Tuna Demirdal, Ümmü Sena Sarı, Salih Atakan Nemli Enfeksiyon Hastalıkları ve Klinik Mikrobiyoloji Kliniği, İzmir Kâtip Çelebi Üniversitesi Atatürk Eğitim Araştırma Hastanesi, İzmir, Türkiye

#### Ozet

Achromobacter cinsi bakteriler, doğada su kaynaklarında ve insan florasında bulunabilen, non-fermantatif, gram negatif basillerdir. İnsanda nadiren enfeksiyon bu mikroorganizmalar çok farklı klinik tablolara neden olmaktadır. Nadiren osteomyelit etkeni olarak görülmektedir. Bu makalede, yaralanma sonrası gelişen, Achromobacterspp,'nin etken olduğu bir kalkaneusosteomyeliti olgusu sunulmuştur. Açık yaraların su kaynakları ile teması sonucu, immün sistemi sağlıklı kişilerde Achromobacter cinsi bakterilerin osteomyelit gibi ciddi enfeksiyonlara neden olabileceği akılda tutulmalıdır.

#### Anahtar Kelimeler

Achromobacter; İmmünokompetan; Kalkaneal osteomyelit

#### Abstract

The Achromobacter species are nonfermenting gram-negative bacilli found in water sources and human flora. These bacteria rarely seen as agents of infection in humans. They cause infections that may present with very different clinical manifestations. In rarely, these microorganisms are agent of osteomyelitis. In this report, calcaneal osteomyelitis with Achromobacter spp. developing after injury has been presented. After open wounds contact with the water, the Achromobacter species that can cause serious infections such as osteomyelitis also in immunocompetent hosts should be taken into consideration.

#### Kevwords

Achromobacter; Calcaneal osteomyelitis; Immunocompetent

DOI: 10.4328/JCAM.3781 Received: 20.07.2015 Accepted: 22.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 680-1 Corresponding Author: Ümmü Sena Sarı, Enfeksiyon Hastalıkları ve Klinik Mikrobiyoloji Kliniği, İzmir Kâtip Çelebi Üniversitesi Atatürk Eğitim Araştırma Hastanesi, Basınsitesi, Karabağlar, İzmir, Türkiye. GSM: +905558742664 E-Mail: dr.kuru\_88@hormail.com

#### Giris

Achromobacter cinsi bakteriler, non-fermantatif, gram negatif basillerdir. İnsan için fırsatçı patojen olan bu mikroorganizmalar, doğada su kaynaklarında ve insan florasında yaygın olarak bulunmaktadır[1]. Achromobacter türleri, pnömoni, bakteriyemi, menenjit, üriner sistem enfeksiyonları, deri ve yumuşak doku enfeksiyonları, endokardit gibi çeşitli enfeksiyonlarda etken olarak rastlanmaktadır[2]. Nadiren de osteomyelit etkeni olarak karşımıza çıkmaktadır [3]. Enfeksiyonların çoğu immün sistemi baskılanmış kişilerde gözlense de, immün sistemi normal kişilerde de Achromobacterenfeksiyonlarına rastlanmaktadır [2].

çalışmada, immün sistemi normal bir hastada Achromobacterspp.'nin neden olduğu kalkaneusosteomyeliti sunulmuştur.

#### Olgu Sunumu

Bilinen bir hastalık öyküsü olmayan 19 yaşında erkek hasta sağ topuğunda sekiz ay önce gelişen delici alet yaralanması sonrası gelişen ağrı şikayeti ile hastanemize başvurdu ve ortopedi kliniğine yatırıldı. Anamnezinde sekiz ay önce sağ ayak tabanına çivi battığı, bu dönemde yaranın kirli su birikintisi ile temas ettiği ve sonrasında da ağrı yakınmasının başladığı öğrenildi.

Fizik muayenesinde sağ ayak plantar yüzde orta hatta şişlik dışında patoloji saptanmadı. Ateş 36C, TA 120/70 mmHg, nabız 84 atım/dk idi. Laboratuvar incelemelerinde; lökosit sayısı 11950/mm3 (%89 nötrofil, %5 lenfosit,%5 monosit), trombosit sayısı 265.000/mm³, hemoglobin 14,9 g/dl, eritrosit sedimantasyon hızı 20 mm/saat, C-reaktif protein 4,32 mg/dl saptandı. Ekstremite MRG' de sağ kalkaneusosteomyeliti ile uyumlu bulgular tespit edildi.

Hasta ortopedi tarafından operasyona alındı ve enfekte doku debride edildi. Operasyon sırasında alınan materyalinin patolojik incelemesine osteomyelit ile uyumlu görünüm izlendi, bakteriyolojik kültüründe Achromobacter spp.üredi. Antibiyotik duyarlılık testlerinde etkenin piperasilin/tazobactama duyarlı olduğu görüldü. Hastaya piperasilin/tazobactam 3x4,5 gr/gün intravenöz tedavi başlandı. Toplam dört hafta intravenöz antibiyotik tedavisi alan, klinik durumu düzelen hasta oral ardışık antibiyotik tedavisine geçilerek taburcu edildi. Oral tedavide etkenin antibiyotik duyarlılık profili göz önünde bulundurularak, siprofloksasin tablet (2x500mg/gün) tercih edildi. İntraveöz ve oral antibiyotik tedavisi toplam süresi sekiz haftaya tamamlandı.

Tedavi sonunda hastanın şikayetlerinin tamamen düzeldiği, kontrol MRG'de bir önceki tetkikle karşılaştırmalı muayenede enfeksiyon bulgularının belirgin gerilediği gözlendi. Hastanın kontrollerinde bir sorunla karşılaşılmadı.

## Tartışma

Achromobacter cinsi bakteriler, non-fermantatif, oksidaz pozitif, katalaz pozitif, aerob, hareketli, gram negatif basillerdir. Gectiğimiz yıllarda aynı ailede yer alan Achromobacter ve Alcaligenes cinsi bakteriler bir hayli taksonomik değişikliğe uğramıştır. Günümüzde Achromobacter cinsi içinde; Achromobacterdenitrificans, Achromobacterpiechaudii, AchromobacterruhlandiveAchromobactexylosoxidanssayılmaktadır. Klinik örneklerden en sık izole edilen tür Achromobacterxylosoxidansolarak karşımıza çıkmaktadır [1]. Yapılan yayınlarda Achromobacter cinsi bakterilerin neden olduğu osteomyelit olgularında A.xylosoxidanstürüsaptanmıştır [3,4]. Olgumuzda teknik sorunlar yüzünden tür tayini yapılamamıştır.

Doğada su kaynaklarında yaygın olarak bulunabilen Achromobacter türlerinden bazıları, insan gastrointestinal sistem ve kulak florasında bulunabilmektedir [1]. Son yıllarda bu mikroorganizmaların neden olduğu hastane kaynaklı enfeksiyonlarda da artış dikkati çekmektedir. Hastane ortamında, diyaliz sıvıları, distile ve deiyonize sular, klorheksidin solüsyonları ve intravenöz sıvılar gibi su ilişkili çevrelerde uzun süre canlı kalabilirler ve nazokomiyalenfeksiyonlara sebep olabilirler [5]. Olgumuzda etkenle olası karşılaşma, şikâyetlerin başlangıcından önce, yaranın kirli su birikintisi ile teması sırasında olduğu tahmin edilmektedir.

Achromobacter türlerinin başlıca izole edildiği klinik örnekler, kan, yara yeri, ürogenital ve solunum sistemi örnekleri, kulak, göz, beyin-omurilik sıvısı ve kemik doku olduğu ifade edilmektedir. Kan dolasımı enfeksiyonlarına daha sık rastlanmaktadır [2]. Acromobacter türleri nadiren osteomyelit etkeni olarak saptanmaktadır [3,4]. Olgumuzda kalkaneusosteomyeliti tanısı kemik doku biyopsisi ile konulmuştur. Klinik örneğin kültüründe Acromobacter spp. üremiştir.

Çok farklı klinik tablolarla seyredebilen Achromobacterenfeksiyonları sıklıkla immünsüpresif hasta grubunda görülmekle birlikte, immün sistemi sağlıklı hastalarda da rastlanmaktadır. Bu mikroorganizmaların etken olduğu enfeksiyonlarimmün direnci bozulmuş yüksek riskli hasta grubunda daha mortal seyreder [2]. Achromobacterenfeksiyonları tedavisinde antibiyotik duyarlılık testlerine göre tedavinin yönlendirilmesi önerilmektedir. Türlere göre değişmekle birlikte genellikle sefalosporin grubu antibiyotiklere dirençlidirler [1]. Bizim olgumuzda etkenin antibiyotik duyarlılık testlerinde penisilinler, sefalosporinler ve aztreonama dirençli, piperasilin tazobaktam, karbapenemler ve kinolonlara duyarlı olduğu saptanmıştır. İntravenöz tedavide piperasilin tazobaktam, idame tedavisinde oral siprofloksasin tercih edilmiş, klinik başarı sağlanmıştır.

Su kaynaklarında yaygın olarak bulunan Achromobacter türleri, sıklıkla immünsüpresif kişilerde görülen, farklı dokularda enfeksiyonlara sebep olan dirençli gram negatif basillerdir. Yaralanma gibi kolaylaştırıcı etken varlığında su kaynakları ile temasın, enfeksiyonun gelişmesinde etkili olduğu düşünülmektedir.

İmmün sistemi sağlıklı bireylerde de bu türlerin osteomyelit gibi ciddi enfeksiyonlara neden olabileceği akılda tutulmalıdır.

#### Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı bevan edilmemiştir.

#### Kaynaklar

- 1. Steinberg JP, Burd EM. Other Gram-Negative and Gram-Variable Bacilli In: Mandell GL, Douglas R, Bennett JE editors. Principles and Practice of Infectious Diseases. 7 ed. New York: Churchill Livingstone 2010:p.3021-2.
- 2. Duggan JM, Goldstein SJ, Chenoweth CE, Kauffman CA, Bradley SF. Achromobacter xylosoxidans bacteremia: report of four cases and review of the literature. Clinical infectious diseases: an official publication of the Infectious Diseases Society of America 1996:23(3):569-76.
- 3. Ozer K, Kankaya Y, Baris R, Bektas CI, Kocer U. Calcaneal osteomyelitis due to Achromobacter xylosoxidans: a case report. Journal of infection and chemotherapy: official journal of the Japan Society of Chemotherapy 2012;18(6):915-8.
- 4. Dubey L, Krasinski K, Hernanz-Schulman M. Osteomyelitis secondary to trauma or infected contiguous soft tissue. The Pediatric infectious disease journal 1988;7(1):26-34.
- 5. Aisenberg G, Rolston KV, Safdar A. Bacteremia caused by Achromobacter and Alcaligenes species in 46 patients with cancer (1989-2003). Cancer 2004:101(9):2134-40.

## How to cite this article:

Demirdal T, Sarı Ü.S, Nemli S.A. A Case Report: Osteomyelitis of Calcaneus Caused by Achromobacter Spp. J Clin Anal Med 2015;6(suppl 5): 680-1.

# 110day of CAM Cas Cas

## A Rare Cause of Phantosmia: Metastatic Small Cell Carcinoma

## Nadir Bir Fantosmi Sebebi: Metastatik Küçük Hücreli Karsinom

Nadir Bir Fantosmi Sebebi / A Rare Cause of Phantosmia

Nihan Coşkun¹, Ali Ercan Altınöz², Behçet Coşar¹¹Department of Psychiatry, Faculty of Medicine, Gazi University, Ankara, ²Psychiatry, Uşak State Hospital, Uşak, Turkey

#### Ozet

Fantosmi olarak bilinen koku varsanıları az anlaşılmış bir fenomendir. Geniş bir ayırıcı tanı yelpazesiyle ilişkili bulunmuştur. Buna rağmen çoğu olgu idiyopatiktir. 70 yaşında metastatik küçük hücreli karsinomunun öncül belirtisi olarak koku varsanısı olan bir erkek hasta, nedensel ilişkiyi açıklayabilmek için sunulmaktadır. Fantosminin kaynağı ve klinik önemi ile ilgili çok az bilgi bulunmaktadır. Kimi zaman olgumuzda olduğu gibi altta yatan metastatik küçük hücreli beyin tümörünün öncül belirtisi olabilmektedir. Bu nedenle organik sebepleri dışlayabilmek için detaylı anamnezin yanı sıra; fizik muayene, nörolojik muayene, rutin laboratuvar ve görüntüleme testlerinin yapılması önerilmektedir.

## Anahtar Kelimeler

Koku Varsanıları; Küçük Hücreli Karsinom; Epilepsi

#### Abstract

Olfactory hallucinations, known as phantosmias, are a poorly understood phenomenon. It has been associated with a wide rage of differential diagnosis. However, most cases are idiopathic. The author's presents a 70-year-old man with olfactory hallucinations as the predominant symptom of the brain metastatic small cell carcinoma in order to clarify the causal relationship. Little is known about the origin and clinical significance of phantosmias. It can even be the predominant symptom of an underlying small cell metastatic brain tumor as presented in our case. Therefore a detailed history of the symptoms along with a neurological and physical examination and routine laboratory and screening tests should be provided in order to exclude any organic causes.

#### Keywords

Olfactory Hallucinations; Small Cell Carcinoma; Epilepsy

DOI: 10.4328/JCAM.3815 Received: 06.08.2015 Accepted: 27.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 682-4 Corresponding Author: Ali Ercan Altinöz, Psychiatry, Uşak Devlet Hastanesi, Uşak, Türkiye.
T.: +90 2762240000 E-Mail: ercanaltinoz@hotmail.com

Phantosmias, also known as olfactory hallucinations, are the olfactory experience in the absence of appropriate stimulation

In a recent population-based study, the prevalence of subjective olfactory dysfunctions have been reported as 4.5% [2]. Among a clinical population with olfactory dysfunctions, the prevalence of parosmia was found to be %34, while the prevalence of phantosmia was %12 [3].

Although most phantosmia cases are idiopathic, many different causes or underlying diseases have been identified for this symptom, including depression, anxiety disorders, posttraumatic stress disorder, schizophrenia, eating disorders, epilepsy, migraine, idiopathic Parkinson's disease, arteriovenous malformations, intracranial hemorrhage, primary and secondary brain tumors, chronic rhinosinusitis [4-10].

Phantosmias could also occur as a form of epileptic aura. Olfactory auras, which are %0.9 of all epileptic auras, are commonly associated with tumors involving the medial temporal lobe and mesial temporal sclerosis. Acharya et all reported that tumor was the most likely etiology in patients with intractable partial epilepsy with olfactory auras, whereas mesial temporal sclerosis was relatively uncommon [9,11].

In addition hallucinations associated with brain tumors have been reported too. Tarachow reported that 96 out of a series of 458 cases (21%) of supratentorial brain tumors had some form of hallucinations. In particular, he reported that olfactory hallucinations, which had been recognized as manifestations of epileptic discharge, were mainly related to lesions in the temporal and frontal lobe [12].

Olfactory hallucinations as the predominant clinical feature of secondary intracranial mass lesions are rare [8].

Here, we report a 70-year-old patient with a brain metastatic small cell carcinoma presenting with olfactory hallucinations as the predominant clinical feature.

#### Case Report

A 70-year-old man was consulted to psychiatry outpatient clinic with a 2 months history of episodic spontaneous fragrant smell of flowers emanating from his chest. He was first presented to neurology outpatient clinic with complaint of headache and carbamazepine treatment was recommended 200 mg per day. On the same day he was consulted to psychiatry outpatient clinic upon this olfactory hallucinations. The patient reported that the smell was of sudden onset without any precipitating factors determined. The smell, which lasted 5-10 minutes, was followed by a feeling of anxiety and palpitation accompanied with a headache and a sensation of numbness and tingling in the head. These symptoms were relieved by drinking water. The episodes were similar in nature and had a frequency of 4-5 times a day. During the episodes there was no alteration in the consciousness, no nausea, no worries about dying or going crazy and no muscle jerking or twitching. Between the episodes he reported a persistent but mild and tolerable headache with no need for analgesia. No previous histories of neurological, psychiatric disorders or trauma have been identified. There was no family history of migraine, seizures or any psychiatric disorder. He was not using any drugs, tobacco products or alcohol. Dur-

ing the last month he kept feeling asleep and according to his relatives he refused to every suggestions or instructions. However he wasn't irritable while he rejected, unless he wasn't insisted. On his mental examination reality testing and insight were found to be partially intact. His attitude was more like negativistic.

Neurological examination was normal. His mini-mental state examination score was 23 but the result might be influenced by the negativistic attitude that was mentioned before. He lost 3 points spelling the word 'world' backwards, 2 points from recalling, 1 point from following a 3-stage command and 1 point from drawing interlocking pentagons.

Laboratory tests, electroencephalography and computerized tomography imaging was requested. The results of electroencephalography were normal. Results of hematological and serum biochemical analyses were within normal limits.

In follow up, one week after starting carbamazepine treatment (200mg/day) his symptoms disappeared. The computerized tomography scan demonstrated a mass lesion with indistinct margins, localized to frontotemporal region surrounded by a significant vasogenic edema [Figure 1]. Thereupon, we referred him to a neurosurgery specialist. After he made an evaluation, he offered an operation in order to excise the mass. Although he rejected the operation because he was totally asymptomatic by the time, he required the surgery upon having left sided central facial paralysis one month later. Then, he went through a surgery by which his mass lesion was excised. The histopathological examination revealed small cell carcinoma.





Figure 1. Computerized Tomography Scan: A mass lesion with indistinct margins. localized to frontotemporal region surrounded by a significant vasogenic edema.

## **Discussion**

Little is known about the origin of phantosmias. Therefore most cases are idiopathic. However, there is a wide range of differential diagnosis of the symptom. Interpretation on hallucinations should be examined in order to reveal psychiatric causes. For example, in depression the hallucination is typically of a foul odour and most of patients believe this to arise from their own bodies. In schizophrenia, patients believe that the smells are being forced on them and take "reasonable" steps to prevent this. In the olfactory reference syndrome the complaint of smell is usually of a true hallucination that the patient believes emanates from himself. This syndrome is characterized by persistent preoccupation about body odour accompanied by shame, embarrassment, significant distress, avoidance behaviour and social isolation [13,14]. In this case the patient believed the smell emanates from himself but he didn't have any delusional

interpretations. He didn't have any other symptom or history of psychiatric disorders and the symptom was sudden and late onset as he was 70 years old. Psychotic symptoms especially the hallucinations in patients with brain tumors may be attributed to an underlying delirium. The patient did not have changes in consciousness or awareness that would have suggested delirium or complex partial seizures.

Olfactory hallucinations have been reported in association with migraine [5] but in these cases the aura is always associated with a headache that fulfills the diagnostic criteria for migraine. In this case although the duration of the hallucinations meets the criteria for migraine auras (5-60 minutes) and the episodes of headache were temporally related to hallucinations, the headache was mild, not lasting at least 4 hours, not prohibiting daily activities and not being associated with physical activity. Olfactory hallucinations could be a manifestation of an epileptic seizure and be called as "epileptic auras". However, not all auras are followed by a seizure [9], as it is true for our patient. Indeed, an aura itself is traditionally called a simple partial seizure without motor symptoms [15] but this phrase is generally used when the case had a confirmed diagnosis of epilepsy and has aura like symptoms temporally unrelated to the seizures.

However hallucinations associated with brain tumors have usually been attributed to an epileptic discharge. The compensatory over-activation of tissue in the nearby brain sensory pathway or a change in global brain excitability after localized, focal compression have been some possible explanations on how tumors could reduce the seizure thresholds.

Anti-epileptic drugs are recommended for individuals with brain tumors who have experienced seizure. In this case the patient was commenced on carbamazepine, and there was a reduction in the frequency and intensity of his symptoms from 2-3 attacks a day at presentation to being asymptomatic within 7 days of commencing this treatment. However there is a case of transient, self-remitting olfactory hallucinations representing a brain tumor [8]. It isn't possible to know if the rapid resolution of this patient's symptoms is a true response to anticonvulsant therapy. It may also be the natural course of this uncommon condition. We could still regard the symptom as an epileptic discharge caused by the brain tumor with the so-called mechanisms, also considering that it has revealed with anti-convulsion treatment.

Most of the studies, which found out that epileptic olfactory hallucinations were related to tumors in the temporal lobe, the epileptic seizures mentioned, were intractable and eliminated by surgery [9,10]. The case is presented with isolated olfactory aura without convulsion or unconsciousness and the aura remitted without surgery.

As a conclusion; olfactory hallucinations are mostly idiopathic and have found to be associated with a broad differential diagnosis. They are rarely seen as the first predominant symptom of a medical illness, including intracranial mass lesions as presented in the case.

### Acknowledgement

This case report was presented as a poster on 18. Annual Meeting and Clinical Education Symposium of Psychiatry Association of Turkey (23-26 April 2014)

Informed Consent: Written informed consent was obtained from patient who participated in this study.

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Ohayon MM. Prevalence of hallucinations and their pathological associations in the general population. Psychiatry Res 2000;97(2-3):153-64.
- 2. Lee WH, Wee JH, Kim D-K, Rhee C-S, Lee CH, Ahn S et al. Prevalence of subjective olfactory dysfunction and its risk factors: Korean national health and nutrition examination survey. PLoS One 2013;8(5):e62725.
- 3. Reden J, Maroldt H, Fritz A, Zahnert T, Hummel T. A study on the prognostic significance of qualitative olfactory dysfunction. Eur Arch Otorhinolaryngol 2007:264(2):139-44
- 4. Lewandowski KE, DePaola J, Camsari GB, Cohen BM, Ongur D. Tactile, olfactory, and gustatory hallucinations in psychotic disorders: a descriptive study. Ann Acad Med Singapore 2009;38(5):383-5.
- 5. Coleman ER, Grosberg BM, Robbins MS. Olfactory hallucinations in primary headache disorders: Case series and literature review. Cephalalgia 2011;31(14):1477-89.
- 6. Landis BN, Burkhard PR. Phantosmias and Parkinson disease. Arch Neurol 2008;65(9):1237-9.
- 7. Epsztein J, Ben-Ari Y, Represa A, Crépel V. Late-onset epileptogenesis and seizure genesis: lessons from models of cerebral ischemia. The Neuroscientist 2008:14(1):78-90
- 8. Lee T-S. Transient and spontaneously-remitting complex hallucinations in a patient with melanoma and brain metastases. Psychosomatics 2010;51(3):267-70.
- 9. Chen C, Shih Y-H, Yen D-J, Lirng J-F, Guo Y-C, Yu H-Y et al. Olfactory Auras in Patients with Temporal Lobe Epilepsy. Epilepsia 2003;44(2):257-60.
- 10. Fried I, Spencer DD, Spencer SS. The anatomy of epileptic auras: focal pathology and surgical outcome. J Neurosurg 1995;83(1):60-6.
- 11. Acharya V, Acharya J, Luders H. Olfactory epileptic auras. Neurology 1998:51(1):56-61.
- 12. Tarachow S. The clinical value of hallucinations in localizing brain tumors. Am J Psychiatry 1941;97(6):1434-42.
- 13. Lochner C, Stein DJ. Olfactory reference syndrome: diagnostic criteria and differential diagnosis. J Postgrad Med 2003;49(4):328-31
- 14. Meats P. Olfactory hallucinations. Br Med J (Clin Res Ed) 1988;296(6622):645. 15. Cavanna AE, Rickards H, Ali F. What makes a simple partial seizure complex? Epilepsy Behav 2011;22(4):651-8.

#### How to cite this article:

Coşkun N, Altınöz A.E, Coşar B. A Rare Cause of Phantosmia: Metastatic Small Cell Carcinoma. J Clin Anal Med 2015;6(suppl 5): 682-4.



## **Cervical Myelopathy Induced** by Ossification of Posterior Longitudinal Ligament

PLLO'ya Bağlı Servikal Miyelopati / Cervical Myelopathy Induced by OPLL

Hidayet Sari<sup>2</sup>, Hamza Sucuoglu<sup>1</sup>, Sarmad Hussain<sup>2</sup>, Ülkü Akarırmak<sup>2</sup> <sup>1</sup>Department of Physical Medicine and Rehabilitation, Private Bagcilar Medicine Center, <sup>2</sup>Department of Physical Medicine and Rehabilitation, Istanbul University, Cerrahpasa Medical Faculty, Istanbul, Turkey

Posterior longitüdinal ligament ossifikasyonu (PLLO), Asya kökenli kişilerde servikal miyelopatinin yaygın nedenlerinden biri olmasına rağmen, diğer ırklarda daha az da olsa görülebilmektedir. PLLO etivolojisi halen net olmamakla birlikte genetik, hormonal ve çevresel faktörlerle ilişkili olduğu kabul edilmektedir. Servikal miyelopati oluşumunda ayrı bir klinik antite olarak karşılaşılan PLLO hiçbir belirti oluşturmayabileceği gibi, ilerleyici nörolojik hasarlara, denge ve yürüme bozukluklarına kadar bir dizi klinik belirtilerle de seyredebilmektedir. PLLO tanısı, tedavi planlaması ve takibi açısından direkt grafi, BT ve MRG önemli yardımcı yöntemlerdir. Tedavide ilerleyici nörolojik bozukluğu olmayan veya hafif miyelopati belirtilerine sahip hastalarda konservatif yaklaşımlar denendiğinde başarılı sonuçlar alınabilmektedir. Ancak, ilerleyici miyelopati bulguları olanlarda cerrahi yaklaşım esas olup, cerrahi yöntemin seçimi hala oldukça tartışmalı bir konudur. Biz de bu amaçla, yaşlı erkek Türk hastamızda servikal miyelopati oluşumuna neden olan PLLO'nun tanı ve tedavisini olgumuz eşliğinde tartışarak sunuyoruz.

#### Anahtar Kelimeler

Servikal Miyelopati; Posterior Longitüdinal Ligament; Ossifikasyon; Rehabilitasyon

#### Abstract

Ossification of Posterior Longitudinal Ligament (OPLL), despite being one of the common causes of cervical myelopathy in Asian subjects it also can be seen less in other races. Although OPLL etiology is still unclear, it is considered to be related to genetic, hormonal and environmental factors. As OPLL; that is encountered as a distinct clinical entity in the formation of cervical myelopathy; may not cause any symptom, it also can be presented with a serie of clinical symptoms like progressive neurological damage and disturbance of balance and gait. Direct X-ray, CT and MRI are important auxiliary method in terms of OPLL diagnosis, treatment planning and follow-up. In patients with no progressive neurological damage or mild symptoms of myelopathy conservative approach can be successful in the treatment. However, surgical approach is the main in patients with progressive myelopathy, the choice of surgical technique is still quite controversial. We therefore offer to discuss the diagnosis and treatment of OPLL that induced cervical myelopathy in our old aged male Turkish patient.

Cervical Myelopathy; Posterior Longitudinal Ligament; Ossification; Rehabilitation

Corresponding Author: Hamza Sucuoglu, Fatih Neighborhood, Maslak Street, Ilke Street, No. 1 Bagcilar, Istanbul, Turkey. GSM: +905056297688 F.: +90 2125784677 E-Mail: hamzasucuoglu@mynet.com.tr

Ossification of the posterior longitudinal ligament (OPLL) of the cervical spine, although reported at 1839 its relation with cervical myelopathy was described at 1960's and in recent years has been considered as a distinct clinical entity [1]. This case is common in Japan and reported rarely in other communities. The etiopathogenesis of OPLL is poorly explained and it is thought to be the result of genetic or geographical factors [2]. Of those presenting with OPLL of the cervical spine, the majority tend to be between 50 and 60 years of age with a male to female ratio of about three to one [1,2]. Cervical myelopathy (CM) is a medulla spinalis injury that results from cervical spondylosis and stenosis caused most often by degenerative causes such as osteophyte, disc calcification and facet hypertrophy. Despite that CM related to OPLL is primarily seen in Asian populations it is also reported in other populations in the literatures [3]. The most common complaint is upper limb weakness and decreased dexterity [4]. Examination consists of a mixture of first and second motor neuron signs. The first motor neuron signs are located under the lesion level and weakness, spasticity, increase in the DTR, pathological reflexes (clonus, Babinski, Hoffman) can be seen [4]. In the diagnosis of CM and OPLL cervical X-ray, computed tomography (CT) and magnetic resonance imaging (MRI) are very helpful. In the treatment of CM cases surgical decompression and / or immobilization is firstly recommended [3]. Conservative treatment methods can be tried in patients with no fast progression, mild myelopathy symptoms or whom refuse undergoing surgery [3,5]. The aim of this case report is to discuss the etiopathogenesis, diagnosis and treatment of OPLL in our Turkish patient with cervical myelopathy due to OPLL.

#### **Case Report**

A 65 aged male patient presented with walking difficulty, balance disturbance and weakness in left arm and leg. The weakness of the left arm started 1.5 year ago then the walking and balance disturbance were added. Patient was admitted to different clinics with these complaints. When the cervical MRI showed that the patient's spinal cord was under pressure he was admitted in our ward for further investigations and treatment. It was learned that our patient had undergone a lumbar disc herniation surgery and left foot drop had developed after that and 23 years ago he had a car traffic accident. In the physical examination; left foot drop and walking with a cane due to balance impairment was observed. Cervical range of motion in all directions was moderately limited and painless. Neurological examination and EMG findings are shown in Table 1. In the laboratory examination hemogram and biochemistry were normal. Cervical X-ray and CT showed that OPLL was extended towards the spinal canal between C3 and C7 levels (Figure 1,2), and spinal compression and myelopathy were seen in the MRI (Figure 3). With these findings the patient was diagnosed with cervical spondylotic myelopathy related to mixed type OPLL. According to Japanese Orthopaedic Association classification [6], our patient was evaluated as grade 1 (JOA Score: 12/17). Accordingly, a conservative treatment was decided to be applied due to the mild myelopathy findings in our patient, symptoms with no rapid progression and the operation refusal by the patient. Treatment of patient: Cervical collar, infrared (500 watt,

Table 1. Neurological Examination and EMG findings

#### Neuorological Examination

Muscle strength\*: Right upper extremity 5/5, left upper extremity 4/5, in lower extremities; left ankle dorsiflexion and toe extension 1/5, others were 5/5

Sensory: Left Cervical (C), C6-C7-C8 and L2-L3-L4-L5 dermatomes were hypoesthetic

DTR: upper extremities were normoactive, left patellar reflex was hyperactive and achilles reflexes bilaterally absent

Pathologic reflexes: Babinski and Hoffman were bilaterally positive.

## EMG Findings

Nerve conduction study is normal. Signs of chronic anterior horn cells damage. SEP has low amplitude.

\*Muscle strength: Evaluated according to Oxford scale.
DTR: Deep tendon reflexes. EMG: Electromyography. SEP: Somatosensory evoked potential.



Figure 1. Lateral cervical x-ray: Mixed type (segmental+continuous) OPLL narrowing the spinal canal between C3-C7.

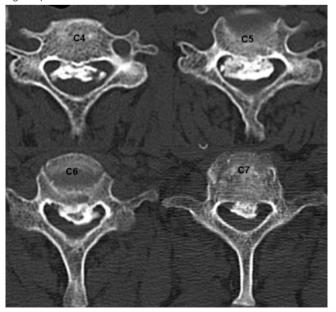


Figure 2. Cervical axial CT: Severe spinal canal narrowing at C3, C4, C5, C6 and C7 vertebral body levels due to OPLL.



Figure 3. Cervical MRI, saggital T2 sequences: Severe spinal canal narrowing, spinal cord thinning and myelomalacic changes due to OPLL between C3-C7.

20 minutes each session, lamp positioned perpendicular to the neck with the patient in sitting position) and ultrasound (frequency of 1MHz, 1.5 W/cm2 intensity, 10 minutes each session) for the neck region, transcutaneous electrical nerve stimulation (TENS) (in conventional mode, frequency of 100Hz, 100µs pulse duration, 20 minutes each session), left upper extremity and the ankle dorsiflexion muscle group electrostimulation (frequency of 20-50Hz, amplitude: 0-100 mA, pulse duration: according to optimal contraction, 15 minutes each session), balance - coordination and walking exercises with a rehabilitation program involving the left upper and lower extremity strengthening exercises were applied. Although there was no significant difference in muscle strength after thirty treatment sessions (5 sessions/ week for 6 weeks), patient had significant improvement in balance and coordination that helped him to walk unaided.

## Discussion

Ossification of the posterior longitudinal ligament (OPLL) is most commonly found in men, in the elderly patients [3]. The incidence of OPLL is 2.4% in the Asian population, and 0.16% in the non-Asian population [7]. While our patient was of a Turkish race despite being an old male. The pathogenesis of OPLL is still unknown. There is some evidence that ligament cells from OPLL patients have osteoblast-like characteristics. Many factors are thought to be the cause of the formation and progression of OPLL like genetic, hormonal, environmental factors and life style but there is no consensus of opinion about this [3]. Although spinal cord injury secondary to road traffic accidents proved to be a predisposing factor for OPLL development, the role of previous traffic accidents in the formation of OPLL is still unclear [8].

OPLL is formed mainly through enchondral ossification, and part of it develops through membranous ossification [3]. In most OPLL cases there is no complaint or mild pain and numbness can be found in the early period. With the progression of neurologic deficits, lower extremity symptoms, such as gait disturbance may appear. OPLL patients show symptoms of myelopathy caused by spinal cord compression rather than ra-

dicular pain due to nerve roots compression [3]. In the examination; at the compression level radicular symptoms and below the compression level upper motor neuron symptoms as DTR increasment, pathological reflexes and spasticity are frequently encountered. Also, walking and balance disturbance forms an important problem [3]. For this purpose, for determining the functional status of patients with cervical myelopathy, a scoring system by the Japanese Orthopaedic Association (JOA Score) is determined [6]. By this scoring system upper and lower extremity motor function, upper and lower extremities and trunk sensory function with bladder and bowel function are evaluated. JOA total score is 0-17. The lower the score the more severe is the deficit. Normal function 16 - 17, Grade 1: 12-15, Grade 2: 8-11, Grade 3: 0-7. According to this scoring system our patient was evaluated as JOA score 12 with grade 1. Direct Xray, CT and MRI are important auxiliary method in terms of CM and OPLL diagnosis, treatment planning and follow-up [3]. Plain radiography is the simplest method to detect OPLL, but it has some limitations especially in the early stages of the disease [3]. OPLL is classified into the following: local type, segmental type, continuous type, and mixed type based on plain radiographic findings. Our patient had a mixed type OPLL according to this classification. Although CT is very successful in terms of viewing osteophytes and calcified discs and evaluating the dimensions of the spinal canal but it does not properly evaluate the spinal cord and cervical roots. In this respect, MRI is highly useful in evaluating spinal cord pressure and the relation between soft tissues and adjuvant bone [4]. In our case, spinal canal narrowing related to mixed type OPLL at the levels between C3 and C7 was obviously seen in x-ray and CT scan while in MRI severe narrowing of the spinal canal at the same level, medulla spinalis thinning and myelomalasic changes were seen. When CM symptoms are mild and not progressive conservative treatment and close follow-up may be good enough. As symptomatic treatments, pain medication, anti-inflammatory drugs, antidepressants, anticonvulsants, and opioid can be applied, and physical therapy such as a brace, are recommended for local stabilization. But when myelopathy findings and neurological symptoms are progressive the surgical treatment is preferred to relieve the spinal cord compression. Surgical approach can be selected based on the degree of myelopathy, the number of involved segments, the location of the primary pathology, the sagittal balance of cervical spine and surgeon's experiences [3]. We applied a conservative treatment to our patient because the myelopathy symptoms are mild and without rapid progression and also the patient did not want a surgical operation. After the treatment our patient became able to walk independently and an improvement in balance and coordination was seen.

#### Conclusion

OPLL, despite being one of the common causes of cervical myelopathy in Asian subjects it can be seen albeit less in other races. OPLL etiology is still unclear. Direct X-ray, CT and MRI are important auxiliary method in terms of OPLL diagnosis and treatment planning. The treatment planning must be done by understanding the advantages and limitations of each method and by considering the neurological condition and the prior medical history of the patient.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Terayama K. Genetic studies on ossification of the posterior ligament of the spine. Spine 1989;14:1184-91.
- 2. Epstein N. The surgical management of ossification of the posterior longitudinal ligament in 51 patients. J Spinal Disord 1993;6:432-55.
- 3. Choi BW, Song KJ, Chang H. Ossification of the posterior longitudinal ligament: A review of literature. Asian Spine J 2011;5(4):267-76.
- 4. Göçmez C, Kamaşak K. Önemli bir tetraparezi nedeni: Servikal spondilotik miyelopati. Dicle Tip Derg 2013;40(3):513-20.
- 5. Sari H, Sucuoglu H, Ornek NI, Misirlioglu TO, Uludag M. The development of upper thoracic myelopathy due to posterior vertebral osteophyte and ossification of ligamentum flavum in patient with diffuse idiopathic skeletal hyperostosis. Turk J Phys Med Rehab 2012;58:342-3.
- 6. Yue WM, Tan SB, Tan MH, Koh DC, Tan CT. The Torg-Pavlov-ratio in cervical spondylotic myelopathy: a comparative study between patients with cervical spondylotic myelopathy and a nonspondylotic, nonmyelopathic population. Spine 2001;26(16):1760-4.
- 7. Tsuyama N. Ossification of the posterior longitudinal ligament of the spine. Clin Orthop Relat Res 1984;184:71-84.
- 8. Gu Y, Chen L, Dong RB, Feng Y, Yang HL, Tang TS. Laminoplasty versus conservative treatment for acute cervical spinal cord injury caused by ossification of the posterior longitudinal ligament after minor trauma. Spine J. 2014;14(2):344-52.

#### How to cite this article:

Sari H, Sucuoglu H, Hussain S, Akarırmak Ü. Cervical Myelopathy Induced by Ossification of Posterior Longitudinal Ligament. J Clin Anal Med 2015;6(suppl 5): 685-8.

## **Ankle Deformity Associated with Torture: A Case Report**

Ayak Bileği Deformitesi / Ankle Deformity

Yunus Güzel<sup>1</sup>, Ali Güleç<sup>2</sup>, Mehmet Elmadağ<sup>3</sup> <sup>1</sup>Department of Orthopaedics and Traumatology, Ordu University, School of Medicine, Campus of Cumhuriyet, Ordu, <sup>2</sup>Department of Orthopaedics and Traumatology, Selçuk University, Selçuklu School of Medicine, Campus of Alaeddin Keykubat, Konya, <sup>3</sup>Department of Orthopaedics and Traumatology, Bezmialem Vakıf University, School of Medicine, İstanbul, Türkiye

İlgili vaka sunumu 2014 yılında Ulusal Ortopedi ve Travmatoloji kongresinde sunulmuştur.

Falaka, akut dönemde ayaklarda cilt altında ve kompartmanlarda ödem ve hemorajiye neden olmaktadır. Şişlik ve ve ağrı dizlere kadar yayılabilir ve enflamatuar süreç bir kaç hafta sonrasında çözülüp yerini, ayak bileğinde elastisite kaybına, ayak ve ayak bileğinde dirençli ağrıya bırakır. Falakadan yıllar sonra ayaklarda ve bacaklarda kronik ağrı yaygındır ancak ileri derecede deformite oluşması nadirdir. Biz bu olgu sunumunda Suriye'de iç savaşta falaka uygulanan bir savaş mağdurunun işkenceye bağlı gelişen ayak bileği deformitesini ve tedavi seçeneği olarak artrodez kullanımını sunacağız.

#### Anahtar Kelimeler

İşkence; Falaka; Ayak Bileği; Deformite

#### Abstract

Falanga causes oedema and haemorrhaging subcutaneously and in compartments of the feet in the acute phase. The inflammatory process leaves a loss of elasticity in the ankle and resistant pain in the foot and ankle. Chronic pain in the feet and legs is common years after falanga but an advanced degree of deformity is rare. The case is presented here of ankle deformity which developed associated with torture applied by falanga in the Syrian civil war, and which was treated by tibiocalcaneal arthrodesis.

#### Keywords

Torture; Falanga; Ankle; Deformity

DOI: 10.4328/JCAM.3818 Corresponding Author: Yunus Güzel, Department of Orthopaedics and Traumatology, Ordu University, School of Medicine, Campus of Cumhuriyet, 52200, Center, Ordu, Türkiye. GSM: +905056996368 E-Mail: dryg@windowslive.com

Falanga is a torture method involving systematically repeated application of blunt trauma to the soles of the feet with rifle butts, wooden poles, iron rods, cables, or sticks. In the acute phase, falanga causes oedema and haemmorrhage in the compartments and below the skin of the feet. Swelling and pain may spread as far as the knees and a few weeks later, rather than being resolved, the inflammatory process leaves a loss of elasticity in the ankle and persistent pain in the foot and ankle [1].

As the most frequently encountered problem following falanga is chronic pain, treatment strategies concentrate on pain management [1-4]. These treatments include shoe modifications such as various orthoses, several forms of physical therapy and methods such as Transcutaneous Electrical Nervous Stimulation (TENS) [1-3] .In the case presented here, surgical treatment was preferred as there was a subtalar joint dislocation and an advanced degree of deformity. To the best of our knowledge, this is the first study to demonstrate a subtalar dislocation due to falanga. In the light of this, the case reported here was evaluated with the literature of ankle deformity associated with torture.

#### Case Report

A 49-year old male presented at our hospital with complaints of loss of movement in the right ankle joint and impaired shape which had developed after he had been subjected to torture 7 months previously, in the Syrian civil war. In the physical examination, there was valgus deformity in the right foot and in the medial of the right ankle there was a 5x5cm granulous wound (Figures 1a and 1b). The patient had no inversion, eversion or plantar flexion movement in the right ankle and dorsiflexion was minimal. The distal pulse was palpable in the right lower extremity. The neurological examination results were normal. In the radiological examination, the calcaneus was displaced laterally and the subtalar joint was seen to be dislocated on direct radiographs (Figures 2a and 2b).

The ankle was entered with an anterolateral incision. As there was a granulous wound in the medial of the ankle, culture was taken because of the possibility of infection. No pathology was seen in the tibiotalar joint. The calcaneus was displaced laterally and there was seen to be no joint connection between the talus and calcaneus. All the debris and capsule adhesions were excised. As the subtalar joint could not be reduced to correct the foot deformity, talus excision together with shortening was performed. Following talectomy, the tibial distal joint surface was removed. Good bleeding bone was exposed and after separating the removed talus from the cartilage, it was then used as graft.

The ankle was prepared at 90°. Tibiocalcaneal arthrodesis was applied with an arthrodesis nail between the calcaneus and the tibia (Figures 3a and 3b). To avoid development of forefoot equinus deformity, a 5mm screw was placed from the navicular bone to the tibia. During the operation, the valgus deformity in the right foot was corrected, the transfer of weight directly from the tibia to the calcaneus was achieved and alignment was seen to be acutely corrected (Figures 4a, 4b and 4c). Discussion



Figure 1. (A) Granulous wound in the inferomedial of the ankle, (B) Image of the valgus deformity



Figures 2. Oblique and lateral radiographs showing the subtalar dislocation (A,B)



Figures 3. Postoperative antero-posterior and lateral radiographs (A,B)

The case is presented here of a 49-year old patient who developed a subtalar joint dislocation following falanga and was treated with ankle arthrodesis.

In the chronic period, apart from subjective complaints such



Figures 4. postoperative image of the acute correction of the deformity (A,B,C)

as persistent pain, cramp and tiredness in most falanga victims, objective clinical findings are not encountered. Although fractures in the phalanx, tarsal bones and calcaneus have been described in studies, bone fractures resulting from falanga are extremely rare [1]. In a study of 59 cases of falanga, only 5 fractures were determined [5]. Although bone fractures are rare, falanga seems to cause the development of advanced level bone deformity.

In the case presented here, the calcaneus was displaced laterally and there was a subtalar dislocation. As this case had been neglected, shortness and tightness had developed in the ankle joint, preventing reduction of the tibiotalar joint. Therefore, the method was selected which would acutely correct the deformity and provide stable fixation. During surgery, talectomy was performed, acutely correcting the deformity and fixation was made with a nail. Thus, the deformity was corrected and a solid fixation was obtained, which would allow weight-bearing in the early period. The most significant problem was that of limb length discrepancy developing after talus excision in a young patient, as after the talus excision there was shortness of 2.5cm in the right leg. The problem was removed by weightbearing with a heel support postoperatively.

To prevent shortness when tibiocalcaneal fusion is applied together with talus excision, it is attempted to apply lengthening at the same time with the Ilizarov method or to remove length difference with structured bone allograft. In a study by Guido et al of 6 patients defined as high risk foot (5 Charcot, 1 infected nail), resection was performed after talectomy and tibiocalcaneal arthrodesis was applied using fibula distal, bone matrix or femoral head allograft [6]. The fixation methods included internal and external fixation methods and the outcomes were found to be satisfactory results.

The foot and ankle are the structures of the body most affected by weight-bearing and which need the greatest stabilisation. In the function of walking, a completely rigid foot serves better than a foot where all the joints are loose. In the case presented here and in other foot and ankle deformities similar to this case, the results were not very pleasing. The surgical treatment applied to the patient presented here will relieve the pain and increase his quality of life. However, the limited ankle range of movement will remain irreversible. There is an evident need for new surgical treatment techniques for the victims of torture.

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Prip K, Persson AL. Clinical findings in men with chronic pain after falanga torture, Clin I Pain 2008:24(2):135-41.
- 2. Amris K, Prip K. Physiotherapy for torture victims with chronic pain. Available treatment at the Rehabilitation Centre for Torture Victims (RCT). Torture
- 3. Lie B, Skjeie H. Torture related injuries a medical challenge. Diagnosis and treatment of falanga victims. Tidsskr Nor Laegeforen 1996;116:1073-5.
- 4. Olsen DR, Montgomery E, Bojholm S, Foldspang A. Prevalent musculo-skeletal pain as a correlate of previous exposure to torture. Scand J Public Health 2006:34:496-503.
- 5. Amris K, Torp-Pedersen S, Rasmussen OV. Long-term consequences of falanga torture - what do we know and what do we need to know? Torture 19(1):33-40. 6. Tellisi N, Fragomen AT, Ilizarov S, Rozbruch SR. Limb salvage reconstruction of the ankle with fusion and simultaneous tibial lengthening using the Ilizarov/Taylor spatial frame. HSS J 2008;4(1):32-42. doi: 10.1007/s11420-007-9073-0.

#### How to cite this article:

Güzel Y, Güleç A, Elmadağ M. Ankle Deformity Associated with Torture: A Case Report, I Clin Anal Med 2015:6(suppl 5): 689-91.



# **Cubital Tunnel Syndrome Due to Synovial Cyst: A Case Report**

## Sinovyal Kistin Neden Olduğu Kubital Tünel Sendromu: Bir Olgu Sunumu

Periferik Tuzak Nöropati / Peripheral Entrapment Neuropathy

Zahir Kizilay¹, Ali Yilmaz¹, Nusret Ok²
¹Neurosurgery Department, Adnan Menderes University Medicine Faculty, Aydın,
²Orthopaedics and Traumatology Department, Pamukkale University Medicine Faculty, Denizli, Turkey

#### Ozet

Sol dirsek ekleminden köken alan bir sinovyal kistin neden olduğu nadir bir ulnar sinir tuzak nöropatisi vakasını sunuyoruz. Elli yedi yaşında erkek bir hasta, 7 aydır sol dirsekte ağrı ve sol elde ilerleyici ve artan bir uyuşukluk ve güçsüzlük şikayetiyle kliniğimize başvurdu. Hastanın yapılan nörolojik muayenesinde 4. ve 5. parmakta güçsüzlük ve his kaybı olduğu tespit edildi. Özellikle hastanın sol dirseği fleksiyon pozisyonuna getirdiğinde Tinel işareti ve Phalen testinin positifti. Elektromiyografi de sol kubital tünel alanında aksonal yaralanma ve tuzak nöropati olduğu tespit edildi. Cerrahi tedavi olarak, total sinovyal kist eksizyonu ve ulnar sinir anterior subkutanöz transpozisyonu yapıldı. Hastanın ağrı şikayeti cerrahiden hemen sonra azaldı. Bu vaka sunumunda, sinovyal kistin neden olduğu kubital tünel sendromunun patofizyolojisi ve bu gibi vakalarda hangi cerrahi tekniğin uygun olabileceği tartıştık.

#### Anahtar Kelimeler

Ulnar Sinir; Kübital Tünel; Sinovyal Kist; Tuzak Nöropati

#### Abstract

We report a rare case of ulnar nerve entrapment caused by a synovial cyst derived from the left elbow joint. A 57-year-old male patient with a seven-month history of pain in his left elbow and a progressive and increasing numbness and weakness complaints in his left hand came to our clinic. Weakness and sensory loss of the 4th and 5th fingers were determined in neurological examination. The results of Tinel's sign and Phalen's Test were positive, especially when his left elbow was flexed. In electromyelography, axonal damage and entrapment neuropathy were determined in the left cubital tunnel area. Total excision of the synovial cyst and ulnar nerve anterior subcutaneous transposition were performed in surgical treatment. The patient's pain decreased immediately after the surgery. In this report, we have discussed the pathopysiology of cubital tunnel syndrome due to synovial cyst and which surgical technique may be suitable as our case report.

#### Keywords

Ulnar Nerve; Cubital Tunnel; Synovial Cyst; Entrapment Neuropathy

DOI: 10.4328/JCAM.3826 Received: 08.08.2015 Accepted: 31.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 692-4 Corresponding Author: Zahir Kizilay, Neurosurgery Department, Adnan Menderes University Medicine Faculty, 09100 Aytepe, Aydin, Turkey.
T.: +90 2564441256 F.: +90 2562148395, +90 2562142040 E-Mail: zahir.kizilay@adu.edu.tr

Ulnar nerve entrapment is the second most-frequent entrapment neuropathy after median nerve entrapment in upper limbs [1]. The ulnar nerve can be compressed at many point, but it is most often observed in the cubital tunnel. The dynamic anatomy and biomechanics of the cubital tunnel affect the ulnar nerve, and result in relative regional ischemia on the nerve. Dynamic and static compression can develop from lesions occupying the space in a limited area such as the cubital tunnel [2]. In this study, we present a rare case of a synovial cyst which was derived from the left elbow joint. The patient's complaints increased when his elbow joint was flexed.

#### Case Report

The case was a 57-year-old male patient with a seven-month history of pain, and progressive and increasing numbness and weakness in his left hand. Weakness and sensory loss of the 4th and 5th fingers were determined in neurological examination. The Tinel's Sign and Phalen's Tests were performed, and his pain was observed as being positive, especially when his left elbow was flexed. The sensory response of the left ulnar nerve could not be detected, the potential amplitude of the compound muscle action was very low, and electromyography showed ulnar nerve entrapment neuropathy with axonal damage at the elbow into the cubital tunnel. Therefore we decided to perform surgery. After obtaining the Informed Patient Consent, we performed an incision in the subcutaneous tissue and opened the cubital tunnel ceiling. During the dissection of the base surface of the cubital tunnel, we observed a cyst which was filled with gelatinous fluid compressing the ulnar nerve (Figure 1). The cyst was totally resected and the ulnar nerve anterior subcutaneous transposition was performed. The patient's pain was relieved immediately after the surgery. Pathological examination was evaluated in favor of a synovial cyst (Figure 2).



Figure 1. Image of the synovial cyst (black arrow) coming from the left elbow joint and located in the cubital tunnel base surface.

## Discussion

Cubital tunnel syndrome is the second most-frequent upper limb entrapment neuropathy after carpal tunnel syndrome [1]. Many ethological factors have been reported in the literature

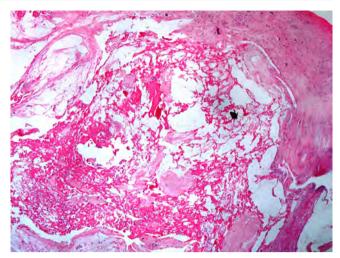


Figure 2. Pathological examination of the synovial cyst with haematoxylin and eosin x 10, synovial membrane and its mucinous content.

such as external traumas in progress, muscles irregulate, subluxation of the ulnar nerve on the medial epicondyle, cubitus valgus, tenosynovitis of rheumatoid arthritis, elbow fractures or dislocations [3]. In addition, soft tissue bulks such as ganglion cysts, synovial chondromatosis and synovial cysts may be considered among rare factors [2]. However, most of the reported cases are idiopathic [4].

The cubital tunnel is defined as an oval tunnel in the elbow through which the ulnar nerve passes. Its upper part is formed by Osborne's Ligament, and its base surface is formed by the elbow joint capsule. The lateral border is formed by the olecranon, and the medial border is formed by the medial epicondyle. The simultaneous effects of elbow movements on the cubital tunnel and the ulnar nerve have been shown in many previous studies. It has been demonstrated in these studies that the cubital tunnel is flattened during the flexion of the elbow, and when the volume of the cubital tunnel decreases by 55%, the pressure in the tunnel increases seven-fold. When contraction of the flexor carpi ulnaris muscle accompanies elbow flexion, the pressure in the tunnel increases more than 20-fold [5, 6]. In addition, the ulnar nerve moves, and is elongated by stretching during the flexion of the elbow with the decrease in the volume of the cubital tunnel [7]. Therefore, it has been claimed that there might be two mechanisms in the pathogenesis of the syndrome that appears due to the trapping of the ulnar nerve in the cubital tunnel in the elbow. These are claimed to be the stretching of the nerve, and the dynamic compression or the repetitive trauma. The dynamic compression, or the repetitive trauma, has been defined as being distal to the cubital tunnel entrapment or being within this entrapment [8].

In our case, the reason for the cubital tunnel syndrome was observed to be the synovial cyst in the elbow; however, the fact that the complaints increased when the elbow of the patient was flexed suggested that it had an effect on the stretching and dynamic compression in pathophysiology. In our study, when the synovial cyst, which is causing the pressure, is related to the elbow joint, the pressure in the cyst may also increase as secondary to the intra-joint pressure with the flexion of the joint, and this may result in the growth of the cyst into the tunnel. As a natural result of this, the cubital tunnel, which has been narrowed in the flexion, may even become narrower, possibly leading to a secondary damage with the dynamic compression of the nerve. In addition, the cyst growing in the flexion may push the nerve upwards from the cubital tunnel ceiling surface, and this may lead to the compression of the nerve as well as suspension of it, thus increasing the tension in the nerve and eventually leading to a secondary injury to the nerve. Actually, this process is similar to the pathological process in radiculopathy formation based on disc herniation. As a result, this increasing compression and tension may destroy the intra-neuronal micro-circulation and axonal transportation and thus lead to clinical findings.

The in situ decompression with mini incision, and endoscopic decompression have both become popular recently, because they are minimally invasive methods. These methods are more suitable for selected cases. On the other hand, when the reason for the cubital tunnel syndrome is a synovial cyst which may originate from the elbow joint, as in our study, these two minimal invasive methods may be insufficient as the pressure on the nerve comes from the base surface of the cubital tunnel, and the patient will eventually need revision surgery. For this reason, we chose the anterior subcutaneous transposition of the ulnar nerve in our case.

Finally, although rare, synovial cysts originating from the elbow joint may be among the ethological reasons for cubital tunnel syndrome. It must be kept in mind that there might be an etiological factor in patients whose complaints increase when the elbow is in flexion. Ulnar nerve damage in the cubital tunnel may be influenced not only by dynamic compression but also by a damage mechanism based on stretching. Anterior subcutaneous transposition is one of the most suitable methods for pathologies originating from the cubital tunnel base surface. We have no financial interest in this manuscript, and we certify that there is no actual or potential conflict of interest in relation to this article.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Asamoto S, Böker DK, Jödicke A. Surgical treatment for ulnar nerve entrapment at the elbow. Neurol Med Chir 2005; 45:240-5.
- 2. Li Y, Lao J. The cubital tunnel syndrome caused by the two synovial cysts. J Plast Reconstr Aesthet Surg 2012; 65(6):827-9.
- 3. Andreisek G, Crook DW, Burg D, Marincek B, Weishaupt D. Peripheral neuropathies of the median, radial, and ulnar nerves: MR imaging features. Radiographics 2006;26(5):1267-87.
- 4. Erol B. Tetik C. Sirin E. The mid-term results of minimal medial epicondylectomy and decompression for cubital tunnel syndrome. Acta Orthop Traumatol Turc 2004:38(5):330-6.
- 5. Apfelberg DB, Larson SJ. Dynamic anatomy of the unlar nerve at the elbow. Plast Reconstr Surg 1973;51.79-81.
- 6. Werner CO, Ohlin P, Elmqvist D. Pressures recorded in ulnar neuropathy. Acta Orthop Scand 1985;56:404-6.
- 7. Gökay NS, Bagatur AE. Subcutaneous anterior transposition of the ulnar nerve in cubital tunnel syndrome. Acta Orthop Traumatol Turc 2012;46(4):243-9.
- 8. Simsek S, Er U, Demirci A, Sorar M. Operative illustrations of the Osborne's ligament. Turk Neurosurg 2011; 21(2):269-70.

#### How to cite this article:

Kizilay Z, Yilmaz A, Ok N. Cubital Tunnel Syndrome Due to Synovial Cyst: A Case Report. J Clin Anal Med 2015;6(suppl 5): 692-4.



## Salmonella Bacteremia: **Pay Attention to Chronic Granulomatous Disease**

Kronik Granülomatöz Hastalık / Chronic Granulomatous Disease

Fatma Deniz Aygün<sup>1</sup>, Fatih Aygün<sup>2</sup>, Serdar Nepesov<sup>1</sup>, Halit Çam<sup>2</sup>, Yıldız Camcıoğlu<sup>1</sup> <sup>1</sup>Çocuk Sağlığı ve Hastalıkları AD, Enfeksiyon Hastalıkları, Klinik İmmünoloji ve Allerji BD, İstanbul Üniversitesi, Cerrahpaşa Tıp Fakültesi,  $^2\mbox{\sc Corrahpaşa}$  Tıp Fakültesi, İstanbul Üniversitesi, Cerrahpaşa Tıp Fakültesi, İstanbul, Türkiye

Süregen yüksek ateş ve septik şok tablosunda kliniğimiz yoğun bakım servisine yatırılarak tetkik ve tedavisi yapılan hastanın sürpriz tanısından söz edilecektir. Hastanın şok tedavisi sürerken alınan kan kültüründe Salmonella enteritidis üremesi ve doğumdan itibaren sık enfeksiyon geçirme öyküsü nedeniyle immün yetersizlik açısından incelendi. Hastanın son 45 günde düşmeyen ateş, lökositoz nedeniyle bir devlet hastanesinde tetkik edildiği, kan ve idrar kültürlerinde etken üretilmediği, viral seroloji ve romatolojik incelemelerin normal olduğu, kemik iliği aspirasyonunda patoloji saptanmadığı öğrenilmiştir. Yapılan immünolojik incelemeler sonucunda NBT testinin düşük olması ve moleküler inceleme sonucu p22phox'da mutasyon saptanarak hastaya otosomal resessif Kronik granulomatöz hastalık tanısı konuldu. Kronik granulomatöz hastalık fagositer sisteme ait NADPH oksidaz eksikliğine bağlı görülen kalıtsal bir primer immün yetersizlikdir. Salmonella enfeksiyonu, çocuklarda çoğunlukla kendini sınırlayan akut gastroenterit belirtileri ile seyreder iken fagosit işlev bozukluğu olan primer immün yetersizlikli hastalarda daha ağır klinik belirtiler ve bakteriyemi ile karşımıza gelebilir.

#### Anahtar Kelimelei

Kronik Granülomatöz Hastalık; Salmonella; Sepsis

#### Abstract

Herein we report a surprising diagnosis of a patient admitting with prolonged high fever and septic shock to our pediatric intensive care unit. She was looked for immunodeficiency because of history of recurrent infections from neonatal period and Salmonella enteritis bacteremia. She was investigated at a local hospital for fever lasting for 45 days, leucocytosis, blood and urine cultures, viral serology, autoimmune markers, bone marrow analysis were all normal. Nitroblue tetrazolium test demonstrated the diagnosis of autosomal recessive chronic granulomatous disease with mutation at p22phox. Chronic granulomatous disease is an immuno deficiency caused by defect in nicotinamid adenine dinucleotide phosphate oxidase enzyme primarily affecting the phagocytes. Salmonella infections are generally mild in children presenting as acute gastroenteritis but can cause bacteremia in patients with altered host defences especially neutrophil dysfunction.

#### Kevwords

Chronic Granulomatous Disease; Salmonella; Sepsis

DOI: 10.4328/JCAM.3834 Corresponding Author: Fatma Deniz Aygün, Çocuk Sağlığı ve Hastalıkları ABD. Çocuk Enfeksiyon BD. İstanbul Üniversitesi Cerrahpaşa Tıp Fakültesi, İstanbul, Türkiye. GSM: +905327868682 F.: +90 2126328633 E-Mail: fdenizaygun@gmail.com

Kronik granülomatöz hastalık [KGH], nikotinamid adenin dinükleotit fosfat [NADPH] oksidaz enzimindeki bozukluk sonucu ortaya çıkan kalıtsal bir primer immun yetersizlik hastalığıdır. NADPH oksidaz enzimi birçok membran proteinleri [gp91phox ve p22phox] ve sitoplazmik proteinlerden [p47phox, p67phox, p40phox, rac] oluşur [1,2]. Bu proteinlerin herhangi birinde oluşan mutasyon sonucu, fagositik sistemin H2O2 ve süperoksit oluşumu engellenmekte ve fagosite edilen mikroorganizmalar öldürülememektedir. Olguların %65'inde görülen gp91phox mutasyonu X'e bağlı resessif olarak kalıtılır iken diğerleri otozomal resesif kalıtım ile oluşmaktadır [3]. Hastalık ilk defa 1954 yılında Janeway ve arkadaşları tarafından tanımlanmış [4], 1959 yılında ise Bridges ve ark. tarafından ise "çocukluk çağının ölümcül granülomatoz hastalığı" şeklinde adlandırmıştır [5]. Katalaz pozitif bakteriler ve mantar enfeksiyonlarına bağlı yineleyen ve yaşamı tehdit eden enfeksiyonlar, abse ve granüloma oluşumu hastalığın başlıca klinik özellikleridir. Bu yazıda düşmeyen ateş ve septik şok nedeniyle yoğun bakım ünitesinde 10 gün entübe kalan ve kan kültüründe Salmonella enteritis üremesi üzerine yapılan incelemeler sonucunda Kronik granulomatöz hastalık tanısı alan 7 yaşında kız hasta, hücre içi bakteriyemisinde nötrofil işlev bozukluğuna ve de primer immün yetersizliklere dikkat çekmek üzere sunulmuştur.

#### Olgu Sunumu

Yüksek ateş nedeni ile kliniğimiz acil servisine getirilen 7 yaşında kız hastanın genel durumu kötü, düşkün, bilinci açıktı. Boy: 120 cm [50.p], kilo: 22 kg [25-50.p] idi. Ateşi:38.60C, kalp tepe atımı 150/dk, kalp sesleri derinden geliyor, belirgin üfürümü yok, S3 duyuluyordu. Tansiyonu 80/42mmHg, periferik nabızları zayıf, kapiller doluş zamanı [KDZ] 4-5 saniyeye uzamıştı. Solunumu takipneik, solunum sesleri doğal, solunum sayısı:33/dakika, oda havasında saturasyonları %85 idi. Bağırsak sesleri artmış, batında yaygın hassasiyet ve gaz vardı. Karaciğer 3 cm kot altı ele geliyordu. Kulak arkasında ve saçlı deride egzematöz deri lezyonları vardı. Düşmeyen ateş sonrasında şok tablosu gelişen hasta sepsis ve olası makrofaj aktivasyon sendromu öntanısı ile yoğun bakıma alındı.

Hastanın 45 gündür düşmeyen ateş, lökositoz ve CRP yüksekliği nedeniyle bir devlet hastanesinde tetkik edildiği, kan ve idrar kültürlerinde etken üremediği, viral seroloji ve romatolojik incelemelerin normal olduğu, kemik iliği aspirasyonunda patoloji saptanmadığı, çekilen batın ve kranial manyetik rezonans [MRI], toraks bilgisayarlı tomografi [BT], Pozitron Emisyon Tomografi + Bilgisayarlı Tomografi [PET-BT] özellik saptanmadığı öğrenilmiştir. Ateş ve enfeksiyon göstergelerindeki yükseklik devam edince ailede ailevi akdeniz ateşi öyküsü olduğu için kolşisin tedavisi başlanmış. Kolşisin altında da ateşi düşmeyince prednizolon eklenmiş. Prednizolonun üçüncü gününde genel durumu bozulup, ishali başlayınca seftriakson tedavisi başlanmış ve kliniğimize sevk edilmişti.

Genel durumunun seftriakson altında kötüleşmesi ve uzun süreli hastane yatışı dikkate alınarak, tüm kültürleri alındıktan sonra hastaya meropenem ve amikasin tedavisi başlandı. Takibinde bilinç durumu kötüleşen ve septik ensefalopati gelişen hasta entübe edilip PCV modunda ventilatöre bağlandı. Eritrosit, albumin ve trombosit desteği verildi. Yoğun bakıma geldiği ilk gün

ve acil serviste alınan toplam 3 kültüründe Salmonella enteritidis üredi. Hastanın ağır seyreden Salmonella enfeksiyonu ve doğumdan itibaren sık enfeksiyon geçirme öyküsü gözönünde tutularak, primer immün yetersizlik hastalıklarından şüphe edildi. İmmunglobulin G, A, M ve E düzeyleri yaşına uygun değerlerdeydi ve hücresel bağışıklığı yansıtan T, B, NK hücrelerin sayısal değerleri düşük bulunmadı. Nötrofillerin işlevleri incelendiğinde ise Nitroblue tetrazolium [NBT] testinin sıfır saptanması üzerine moleküler inceleme istendi. p22phox eksikliği saptanan hastaya otosomal resessif Kronik granulomatöz hastalık tanısı konuldu.

Kronik Granulomatöz Hastalık tanısı konulan hastaya yoğun bakımda tedavisi tamamlandıktan sonra çıktıkta itrakanazol ve trimetoprim-sulfametaksazol profilaksisi başlandı ve fakültemiz Çocuk İmmunuloji Bilim dalı tarafından izleme alındı.

#### Tartışma

NADPH oksidaz enzimi, hücre membranında sitokrom-b558 [gp91phox ve p22phox bileşeni] ve sitoplazmada p67phox, p47phox, p40phox ve Rac 1/2'den gibi birçok proteinden oluşur. Kronik granülomatöz hastalık, doğal immun sistemin bir elemanı olan bu enzim sistemindeki bozukluğa bağlı gelişen fagositer sistem hastalıklarından biri olup sıklığı 125.000 canlı doğumda 1 olduğu kabul edilmektedir. Mikroorganizmaların fagositik hücreler tarafından yutulduktan sonra solunumsal patlama ile hücre içinde öldürülememesi sonucu granüloma oluşumuna yol açmaktadır. Hastalık belirtileri sıklıkla erken çocukluk döneminde başlar ve ortalama tanı yaşı 2.5-3 yaş olarak bildirilmiştir [1]. Hastalık erişkin dönemine kadar gecikebilir. Hastalığın en sık [%65 görülen tipi X'e bağlı kalıtılır ve gp91phox geninde [Xp21.1] mutasyonu sonucunda oluşur. Mutasyon olan erkek çocuklarda hastalık diğer otozomal resessif geçişli tiplerine göre daha ağır seyreder ve yaşamın ilk yıllarında hayatı tehdit eden katalaz pozitif mikroorganizmaların neden olduğu enfeksiyonlar gelişebilir [3]. Otozomal resessif geçişli tiplerden p22phox %5, p67phox %5 ve p47phox mutasyonu ise %25 oranında görülür, bu hastalık tiplerinde hasta erişkin yaşa kadar tanı almayabilir. Ülkemizde 89 hastanın irdelenmesinde % 22.5 oranında p22phox mutasyonuna rastlanmıştır [6]. Bizim olgumuzun da genetik incelemesi p22phox mutasyonu ile uyumlu saptanmıştır.

Pnömoni, menenjit, septisemi, abse oluşumu, supuratif adenit, osteomyelit, sellulit gibi enfeksiyonlar hastalığın başlıca klinik özellikleridir. Enfeksiyon sıklığı Martire ve arkadaşları tarafından 0.15-0.3/yıl oranında bildirilmiştir [7]. Staphylococcus aureus ve Aspergillus türleri gibi katalaz üreten mikroorganizmalarla enfeksiyonlar en sık görülmekle birlikte Burkholderia cepacia, Klebsiella pneumoniae ve Serratia marcescens diğer rastlanan mikroorganizmalardır. Salmonella, Bacille Calmette-Guerin [BCG] ve Mycobacterium tuberculosis ise Avrupa'da enfeksiyon etkeni olarak daha sık görülmektedir [3]. Mantar enfeksiyonları %20 oranında görülür ve Aspergillus türleri en sık görülen fungal ajandır ve bazan hastalığın ilk bulgusu olabilir.

Salmonella gram negatif, hareketli, fakultatif anaerob intraselüler bir mikroorganizmadır. Salmonella enfeksiyonları çocuklarda asemptomatik gastrointestinal sistem taşıyıcılığından kendini sınırlayan akut gastroenterite kadar farklı klinik bulgu verebilirler. İmmün yetersizlikli hastalarda ise menenjit, osteomyelit ve beyin absesi gibi daha ağır enfeksiyonlar gelişebilir. Salmonella gastroenteriti sonucu bakteriyemi gelişme oranı ise %5'den azdır. Bu nedenle ağır salmonella enfeksiyonu geçirmekte olan hastalar primer immün yetersizlik açısından incelenmelidir.

Olgumuzun 3 kez alınan kan kültüründe hep Salmonella üremesi ve sık enfeksiyon geçirme öyküsü, primer immun yetmezlikten şüphe edilmesine neden olmuştur.

Hastalığın tanısında nitroblue tetrazolium reduksiyon testi [NBT] ve dihidrodamin-123 [DHR] oksidasyon yöntemi kullanılır. NBT testi sıfır olan olgumuzun tanısı DHR testi ile de doğrulanmıştır [8].

Hastaya tanı konulduktan sonra hemen antimikrobiyal proflaksi başlanmıştır. KGH'li olgularda en sık görülen mikroorganizmalara karşı etkili olduğundan Trimetoprim-sulfametoksazol antibakteriyel proflakside tercih edilmektedir, hastava 5 mg/kg/gün iki dozda kullanmak üzere başlanmıştır. Antifungal proflakside ise yan etkisinin az olması ve Aspegillus'lara karşı etkinliği nedeniyle itrakonazol önerilmiştir.

KGH'lu hastalarda granulom oluşumu [1] ve otoimmünite görülebilir. Olgumuzda herhangi granulom oluşmamış ve otoimmunite gelişmemiştir.

Salmonella enfeksiyonu, çocuklarda çoğunlukla kendini sınırlayan akut gastroenterit belirtileri ile seyreder iken fagosit işlev bozukluğu olan primer immün yetersizlikli hastalarda bakteriyemi ve ciddi fokal enfeksiyon gibi daha ağır klinik belirtiler görülebilir. Kan kültüründe Salmonella üremesi olan hastalar immun yetersizlik açısından değerlendirilmeli ve mutlaka fagosit işlevleri incelemelidir.

#### Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı beyan edilmemiştir.

#### Kaynaklar

- 1. Holland SM. Chronic granulomatous disease. Clin Rev Allergy Immunol 2010; 38(1): 3-10.
- 2. De Oliveira EB. Bustamante I. Newburger PE. Condino-Neto A. The human NADPH oxidase: primary and secondary defects impairing the respiratory burst function and the microbicidal ability of phagocytes. Scand  $\boldsymbol{J}$  Immunol 2011;73(5):420-7
- 3. Segal BH, Leto TL, Gallin JI, Malech HL, Holland SM. Genetic, biochemical, and clinical features of chronic granulomatous disease. Medicine 2000 ;79(3):170-
- 4. Janeway CA, Craig J, Davison M, Doroney W, Gitlin D, Sullivan JC. Hypergammaglobulinemia associated with severe, recurrent, and chronic non-specific infection. Am J Dis Child 1954;88(1):388-92.
- 5. Bridges RA, Berendes H, Good RA, A fatal granulomatous disease of childhood: the clinical, pathological, and laboratory features of a new syndrome. AMA J Dis Child 1959;97(4):387-408.
- 6. Köker MY, Camcıoğlu Y, van Leeuwen K, Kılıç SŞ, Barlan I, Yılmaz M, et al. Clinical, functional, and genetic characterization of chronic granulomatous disease in 89 Turkish patients. J Allergy Clin Immunol 2013;132(5):1156-63.
- 7. Martire B, Rondelli R, Soresina A, Pignata C, Broccoletti T, Finocchi A, et al. Clinical features, long-term follow-up and outcome of a large cohort of patients with Chronic Granulomatous Disease: an Italian multicenter study. Clin Immunol 2008;126(2):155-64.
- 8. De Ravin SS, Naumann N, Cowen EW, Friend I, Hilligoss D, Marguesen M, et al. Chronic granulomatous disease as a risk factor for autoimmune disease. J Allergy Clin Immunol 2008;122(6):1097-103.

## How to cite this article:

Aygün F.D, Aygün F, Nepesov S, Çam H, Camcıoğlu Y. Salmonella Bacteremia: Pay Attention to Chronic Granulomatous Disease. J Clin Anal Med 2015;6(suppl 5):



## Non-Operative Management of Isolated Pneumoperitoneum Due to Severe Blunt Abdominal Trauma

## Şiddetli Künt Karın Travmasına Bağlı İzole Pnömoperitonyumun Cerrahi Dışı Yönetimi

Künt Karın Travmasında İzole Pnömoperitonyum / Isolated Pneumoperitoneum in Blunt Abdominal Trauma

Murat Özgür Kılıç<sup>1</sup>, Mikdat Bozer<sup>1</sup>, Ahmet Türkan<sup>1</sup>, Yeliz Kılıç<sup>2</sup>

Department of Surgery, Faculty of Medicine, Turgut Ozal University,

Clinic of Anesthesiology and Reanimation, Dr. Abdurrahman Yurtarslan Oncology Training and Research Hospital, Ankara, Turkey

An abstract of this article was presented as a poster in 19. National Surgery Congress, 2014.

#### Özet

Künt karın travmasının cerrahi dışı yönetimi hemodinamik olarak stabil hastalarda tercih edilen tedavi yöntemidir. Cerrahi dışı yönetimin sonuçları, içi boş organ yaralanmasına göre, karaciğer ve dalak gibi izole solid organ yaralanmalarında daha başarılıdır. Bu yaklaşımda, hem hastanın klinik seyri hem de bilgisayarlı tomografi bulguları önemli bir rol oynar. Künt karın travmasında serbest peritoneal hava klinisyenler için cerrahi bir zorluk olabilir çünki genellikle içi boş organ perforasyonu için önemli bir radyolojik işarettir. Burada, ciddi bir künt karın travmasına maruz kalan genç bir erkekte, bilgisayarlı tomografide saptanan ve cerrahisiz yönetilen, izole pnömoperitonyum olgusu sunuyoruz. Amacımız, künt karın travmasında, diğer radyolojik bulguların yokluğunda, pnömoperitonum varlığının yönetim sorunlarına cerrahların ilgisini çekmektir.

#### Anahtar Kelimele

Türkçe anahtar kelimeleri lütfen secretary@jcam.com.tr gönderiniz

#### Abstract

Nonoperative management of blunt abdominal trauma is the treatment of choice for hemodynamically stable patients. The results of nonoperative management are more successful in isolated solid organ injuries such as the liver and spleen than hollow viscus injury. In this approach, both the clinical course of the patient and the computed tomography findings play an important role. Isolated pneumoperitoneum in blunt abdominal trauma may be a surgical challenge for clinicians because it is usually a significant radiological sign for hallow viscus perforations. Here, we report a case of isolated pneumoperitoneum detected on computed tomography and managed non-surgically, in a young man suffered from a severe blunt abdominal trauma. Our aim is to attract the attention of surgeons to the management problems of the presence of pneumoperitoneum in the absence of other radiological findings in blunt abdominal trauma.

#### Kevwords

ingilizce keywords lütfen secretary@jcam.com.tr gönderiniz

DOI: 10.4328/JCAM.3847 Received: 21.08.2015 Accepted: 02.08.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 698-700 Corresponding Author: Ahmet Türkan, Department of General Surgery, Faculty of Medicine, Turgut Ozal University, Ankara, Turkey, Gsm: +905055951637 E-Mail: draturkan@gmail.com

In recent years, there have been significant changes in the approach to blunt abdominal trauma. Conservative approaches have been increasingly used according to the improvements in radiological methods such as ultrasonography (US) and computed tomography (CT), interventional radiology, and critical care [1]. Although many cases with blunt abdominal injury can be managed non-surgically, this decision may not be always received easily. In some cases with blunt injury, free peritoneal air detected by radiological methods, may be the single imaging finding and may not always indicate perforation of hollow viscus. In this condition, such patients should be evaluated as a whole with repeated physical examinations, laboratory findings and advanced radiological tests before taking a decision of emergent surgery.

#### Case Report

A 42-year-old man was brought to emergency room with severe crush injury on the upper half of his body caused by an accident during the repair of lift. The patient was hemodynamically stable and the laboratory findings were all within normal limits. On clinical examination, the abdomen was soft and painless. Chest x-ray revealed pneumothorax, 1 cm in size, in both hemithorax. On abdominal plain radiography, peritoneal free air was not determined. US showed no abnormality. Computed tomography (CT) visualized peritoneal free air in right subphrenic space in the absence of free fluid and solid organ injury (figure). Additionally, there was no evidence of traumatic rupture of the diaphragm. However, fractures were detected in three ribs with minimal pneumothorax. The patient had also minor fractures in lombar 1-4 vertebras and left scapula. Pneumoperitoneum was thought to derive from pneumothorax. The patient was closely monitored at the intensive care unit without any surgical intervention. The subsequent course was uneventful and the patient was discharged on tenth day.

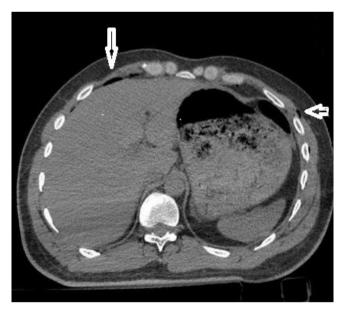


Figure. Tomographic image of free subdiaphragmatic peritoneal air (arrows)

#### Discussion

In blunt trauma, the clinical findings are usually not reliable. The

management of blunt abdominal trauma is quite difficult and requires much attention. CT scan has become the diagnostic modality of choice in the management of patients with blunt abdominal injury [2,3]. Use of CT scan has dramatically changed the methods for diagnosing blunt abdominal trauma and refined the surgeon's decisions. It is mainly indicated for hemodynamically stable patients with equivocal findings on physical examination. In addition, drugs, alcohol, head trauma and spinal cord injury are the other endications for CT use [3]. CT is very useful in defining the severity of solid organ injury such as liver, spleen and kidney and guiding the NOM and decisions for laparotomy. In blunt abdominal trauma, gastrointestinal perforation is one of the most common causes of intraperitoneal free air. These perforations are emergency conditions requiring an early recognition and a timely surgical treatment. CT is the best imaging method of detecting it but no clear consensus exists on its role especially in hollow viscus and mesenteric injuries. Although the presence of pneumoperitoneum in blunt abdominal trauma was a certain indication of laparotomy previously, effective and commonly use of CT in blunt abdominal trauma enabled surgeons to select patients for conservative treatment, and therefore the rate of conservative approaches has began to increase in recent years. Neverthless, missed hollow viscus injuries still remain a significant problem, as they considerably increase morbidity and mortality in patients with multiply trauma. It should be noted here that pneumoperitoneum detected by CT scan, is not always result from perforation of the gastrointestinal tract and thus may not require laparotomy [4]. Because only 45% of patients with gastrointestinal perforation have free peritoneal air on imaging methods [5]. This condition is called as spontaneous, nonsurgical, or idiopathic pneumoperitoneum, and usually poses a dilemma to the surgeon. Mechanical ventilation, cardiopulmonary resuscitation and pneumomediastinum are the other causes of free intraperitoneal air [6]. Hamilton et al. also reported that pneumoperitoneum can be developed secondary to dissection of interstitial air from the chest [7]. Similarly, in our case, free intraperitoneal gas was thought to derive from pneumothorax, according to the normal clinical findings and the absence of radiological findings indicating any abdominal organ injury.

In conclusion, the presence of free intraperitoneal air in blunt abdominal trauma without a detectable solid organ injury is a significant surgical dilemma for most surgeons. It should be kept in mind that free peritoneal gas may occur as a result of extra-abdominal conditions as was in our case. Although surgery is the main treatment in the presence of pneumoperitoneum previously, hemodynamically stable patients with normal abdominal examination findings can be managed conservatively in blunt abdominal trauma, with closed monitoring.

## Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Ozoğul B. Kısaoğlu A. Avdınlı B. Oztürk G. Bayramoğlu A. Sarıtemur M et al. Nonoperative management (NOM) of blunt hepatic trauma: 80 cases. Ulus Travma Acil Cerrahi Derg 2014:20(2):97-100.
- 2. Thorelius L. Emergency real-time contrast-enhanced ultrasonography for the detection of solid organ injuries. Eur Radiol Suppl 2007;17(Suppl.6):107-12.
- 3. Peitzman AB, Makaroun MS, Slasky BS, Ritter P. Prospective study of com-

puted tomography in initial management of blunt abdominal trauma. J Trauma 1986;26(7):585–92.

- 4. Marek AP, Deisler RF, Sutherland JB, Punjabi G, Portillo A, Krook J et al. CT scandetected pneumoperitoneum: An unreliable predictor of intra-abdominal injury in blunt trauma. Injury 2014;45(1):116-21.
- 5. Kumar A, Muir MT, Cohn SM, Salhanick MA, Lankford DB, Katabathina VS. The etiology of pneumoperitoneum in the 21st century. J Trauma Acute Care Surg 2012;73(3):542-8.
- 6. Mularski RA, Sippel JM, Osborne ML. Pneumoperitoneum: a review of nonsurgical causes. Crit Care Med 2000;28(7):2638-44.
- 7. Hamilton P, Rizoli S, McLellan B, Murphy J. Significance of intra-abdominal extraluminal air detected by CT scan in blunt abdominal trauma. J Trauma 1995;39(2):331-3.

#### How to cite this article:

Kılıç M.Ö, Bozer M, Türkan A, Kılıç Y. Non-Operative Management of Isolated Pneumoperitoneum Due to Severe Blunt Abdominal Trauma. J Clin Anal Med 2015;6(suppl 5): 698-700.



# **Ruptured Ovarian Dermoid Cyst Mimicking Peritoneal Carcinomatosis: CT and MRI**

Peritoneal Karsinomatozis, Rüptüre Ovaryan Dermoid Kist / Peritoneal Carcinomatosis, Ruptured Ovarian Dermoid Cy		
Gurcan Erbay, Merve Ozen, Elif Karad Radyoloji Anabilim Dalı, Başkent Üniversitesi Tıp Fakültesi, Adana, Türki		

#### Özet

Ovaryan dermoid kistin spontan rüptürü ve kist içeriğinin kronik abdominal sızması granülomatoz peritonite neden olan çok nadir bir durumdur, peritoneal karsinomatozis ile karışabilir. Burada peritoneal karsinomatozisi taklit eden radyolojik bulgularıyla kronik granülomatozis nedeni olan rüptüre dermoid kist olgusunu sunduk.

#### Anahtar Kelimeler

Dermoid Kist; Over; Peritoneal Karsinomatozis; Rüptür

## Abstract

Spontaneous rupture of ovarian dermoid cyst and chronic abdominal spillage of its content is a very uncommon condition, which causes granulomatous peritonitis and can be confused with peritoneal carcinomatosis. Here is presented such a case of ruptured dermoid cyst causing chronic granulomatous peritonitis with radiologic findings mimicking peritoneal carcinomatosis.

Dermoid Cyst; Ovarian; Peritoneal Carcinomatosis; Rupture

DOI: 10.4328/JCAM.3684 Corresponding Author: Elif Karadeli, Radyoloji Anabilim Dalı, Başkent Üniversitesi Tıp Fakültesi, 01250, Yüreğir, Adana, Türkiye. T.: +90 3223272727 E-Mail: elifkaradeli @gmail.com

#### Introduction

Ovarian dermoid cysts are common lesions, accounting for up to 10-25% of all ovarian neoplasms[1]. Reported complications include torsion (16%), rupture (1-4%), malignant transformation (1-2%), infection (1%), invasion into adjacent viscera and autoimmune hemolytic anemia (<1%). The spontaneous rupture into adjacent viscera is one of the least common complications [2]. Here is presented a case of ruptured ovarian dermoid cyst with granulomatous peritonitis, with radiologic findings mimicking peritoneal carcinomatosis.

#### **Case Report**

A 54 year-old lady was referred to our clinic for further assessment of mild abdominal pain, fatigue and intra-abdominal free fluid. The patient had no operation history. Oral informed consent was obtained from the patient. Abdominal computed tomography (CT) revealed intra-abdominal free fluid and diffuse peritoneal infiltrative lesions (with 3.5 cm thickness), which is considered as "omental cake" (Figure 1).

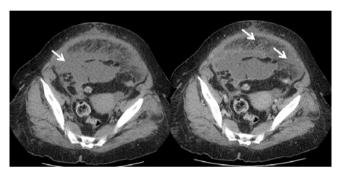


Figure 1. Precontrast and postcontrast axial computed tomography images show intraperitoneal free fluid and 'omental cake' appearance.

No calcification or cystic lesions were found. According to these findings, our first impression as diagnosis was peritoneal carcinomatosis. Laboratory work-up revealed high serum CA125, carcinoembryonic antigen CEA and CA19-9 levels (109 U/mL, 12,1 ng/mL and 23.576 U/mL, respectively). She had leukocytosis (14,20 K/mm3) with a serum CRP level of 107,30 mg/L. MRI was performed for differential diagnosis: an amorphous, thin-walled, cyst-like right adnexal lesion of 9.8x7.0x5.5 cm was detected. The margins of lesion were discontinuous. Additionally restricted diffusion of omentum on diffusion weighted images (DWI), some fat - fluid levels on T2WI and foamy appearance of fat signals were found on MRI (Figure 2,3).

High lymphocyte count was detected in the peritoneal fluid on peritoneal irrigation. Surgery revealed a ruptured right ovarian dermoid cyst, peritonitis and bowel adhesions. Omentectomy, total abdominal hysterectomy and salpingo-oophorectomy were performed. The frozen biopsy sections of peritoneum and right ovary were reported to be benign. Early postoperative course and 18-month follow-up of the patient was eventless.

#### Discussion

The typical radiologic finding of dermoid cyst is intratumoral fat. The most common ultrasound finding is a cystic mass with an echogenic tubercule due to hair, teeth, and fat [3]. At CT, intratumoral fat shows negative attenuation [4]. At MRI, intratumoral fat shows hyperintense on T1-weighted images, and

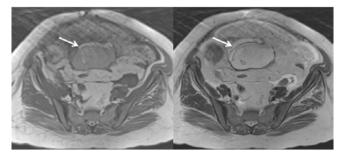


Figure 2. Axial T1 and T2 weighted images demonstrate amorphous, thin-walled, cyst-like right adnexal lesion. In addition, there are fat - fluid levels and foamy appearance.

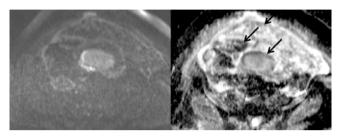


Figure 3. On diffusion weighted magnetic resonance images and apparent diffusion coefficient maps, there is the restricted diffusion as a pitfall.

drop on fat-saturated T1-weighted images. In addition to intratumoral fat, calcification is also commonly seen in ovarian dermoid cysts, but it does not always indicate ovarian dermoid cyst. Therefore, the presence of intratumoral fat is important in the diagnosis of ovarian dermoid cysts [3,5]

The ovarian dermoid cysts have keratinoid content, therefore, they have been shown to exhibit lower ADC values than any other benign or malignant adnexal lesion, a characteristic that may help diagnose lesions with a paucity of fat [6]. We detected restricted diffusion on ADC maps in the both lesion and omental areas, therefore, we thought malignancy.

Spontaneous rupture is an extremely rare complication of dermoid cyst (also known as mature cystic teratoma). Because, it usually has a thick capsule. The reported causes of rupture are torsion with infarction of the tumor, infection, malignancy and rapid growth of the cyst, direct trauma, or prolonged pressure during pregnancy [7]. At ultrasound, CT and MRI, the integrity of the tumor wall should be carefully evaluated for accurate diagnosis of a ruptured ovarian dermoid cyst [8]. Similar to these findings, the integrity of the tumor wall in our case was distrupted.

There has been two possible clinic presentations for ruptured dermoid cysts reported in the literature. First presentation is spillage of tumor contents into the abdominal cavity due to rupture, it can lead severe acute chemical peritonitis. Second, chronic granulomatous peritonitis due to chronic cyst content leak is characterized by multiple small white peritoneal lesions, dense adhesions, and ascites mimicking carcinomatosis or tuberculous peritonitis [9].

Chronic granulomatous peritonitis usually has unusual radiologic findings: ascites and omental infiltration. These findings simulate carcinomatous or tuberculous peritonitis. Ovarian and gastrointestinal system malignancies usually manifest with ascites and peritoneal carcinomatosis.

Serum tumor markers (CA19-9, CEA, CA 125) tend to be high in gastrointestinal system lesions. In our case, high tumor mark-

ers, ascites, "omental cake" appearance were suggestive of a malignant tumor.

As a very rare entity, chronic granulomatous peritonitis due to ruptured ovarian dermoid cyst can be easily confused with peritoneal carcinomatosis. Mild chronic abdominal pain, a very thin cyst wall remnant, fat-fluid levels, and foamy fat signals on MRI are important clues for dermoid cyst rupture and chronic spillage. Hence, MRI is highly important to make the differential diagnosis.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Tandon A, Gulleria K, Gupta S, Goel S, Bhargava SK, Vaid NB. Mature ovarian dermoid cyst invading the urinary bladder. Ultrasound Obstet Gynecol 2010;35(6):751-3.
- 2. Godara R, Karwasra RK, Garg P, Sharma N. Pilimiction. A diagnostic symptom of ovarian dermoid cyst. Internet J Gynecol Obstet 2006;6(1):62-9.
- 3. Outwater EK, Siegelman ES, Hunt JL. Ovarian teratomas: tumor types and imaging characteristics. Radiographics 2001;21(2):475-90.
- 4. Buy JN, Ghossain MA, Moss AA, Bazot M, Doucet M, Hugol D, et al. Cystic teratoma of the ovary: CT detection. Radiology 1989;171(3):697-701.
- 5. Guinet C, Ghossain MA, Buy JN, Malbec L, Hugol D, Truc JB, et al. Mature cystic teratomas of the ovary: CT and MR findings. Eur J Radiol 1995;20(2):137-43.
- 6. Nakayama T, Yoshimitsu K, Irie H, Aibe H, Tajima T, Nishie A, et al. Diffusion weighted echo-planar MR imaging and ADC mapping in the differential diagnosis of ovarian cystic masses: usefulness of detecting keratinoid substances in mature cystic teratomas. J Magn Reson Imaging 2005;22(2):271-8.
- 7. Nader R, Thubert T, Deffieux X, Laveaucoupet J, Ssi-Yan-Kai G. Delivery induced intraperitoneal rupture of a cystic ovarian teratoma and associated chronic chemical peritonitis. Case Reports in Radiology 2014;2014:189409. doi:10.1155/2014/189409.
- 8. Fibus TF. Intraperitoneal rupture of a benign cystic ovarian teratoma: findings at CT and MR imaging. AJR Am J Roentgenol 2000;174(1):261-2.
- 9. Pantoja E, Noy MA, Axtmayer RW, Colon FE, Pelegrina I. Ovarian dermoids and their complications: comprehensive historical review. Obstet Gynecol Surg 1975:30:1-20.

#### How to cite this article:

Erbay G, Ozen M, Karadeli E. Ruptured Ovarian Dermoid Cyst Mimicking Peritoneal Carcinomatosis: CT and MRI. J Clin Anal Med 2015;6(suppl 5): 701-3.

# 110 da A Solumino Carresto Car

## Aphasia, Just a Neurological Disorder?

## Afazi Yalnızca Nörolojik bir Bozukluk Mudur?

Afazi / Aphasia

Mehmet Tamer Özdemir¹, Eray Atalay¹, Şevki Hakan Eren², Bilge kağan Tur¹, Ferhat Omurca¹, Gül Gürsoy¹ ¹İç Hastalıkları AD., Kafkas Üniversitesi Tıp Fakültesi, Kars, ²Acil Tıp AD., Cumhuriyet Üniversitesi Tıp Fakültesi, Sivas, Türkiye

#### Ozet

Hashimoto ensefalopatisi (HE), otoimmün tiroidit ile ilişkili nadir görülen bir hastalıktır. Etiyolojisi tam olarak anlaşılamamıştır. Serum antitiroid antikorları HE'de yüksek seviyelerde görülür. Karakteristik olarak HE Bakteriyel veya viral enfeksiyonların yokluğunda, bilişsel bozukluk, psikiyatrik ve nörolojik belirtiler ile birlikte görülen otoimmun etyolojili bir tablodur. Steroid tedavisine duyarlı bir ensefalopatidir. Altmış yaşında erkek hasta, yaklaşık 9 aydır devam eden unutkanlık ve son 2 gündür olan konuşma kaybı nedeni ile hastaneye kabul edildi. Tiroid fonksiyon testlerinde ileri derece hipotroidi tespit edilmesi üzerine yapılan Elektroensefalografi ve MR görüntülemeleri HE ile uyumlu bulundu. Hashimoto ensefalopatisi nadir görülen ve yeterince tanınmayan bir durum olduğundan diğer nedenler dışlandıktan sonra ayırıcı tanıda düşünülmesi gereken bir hastalıktır. Hashimoto ensefalopatisi doğru tanısı bilişsel ve klinik sunumların farkındalığı ile konulabilir.

#### Anahtar Kelimeler

Hashimoto Ensefalopatisi; Afazi; Nörolojik Bozukluk

#### Abstract

Hashimoto's encephalopathy (HE) is a rare disorder associated with autoimmune thyroiditis. Etiology of HE is not completely understood. High levels of serum antithyroid antibodies are seen in HE. Presentation with otoimmune thyroiditis, cognitive impairment, psychiatric and neurologic symptoms and absence of bacterial or viral enfections are characteristics of HE. HE is a steroid responsive encephalopathy. 60 years old male patient admitted to hospital with forget fulness continuing for 9 months and speech loss starting 2 days ago. Strong positivity of antithyroid antibodies increases the odds for HE. Thyroid function tests showed severe hypothyroidism. Electroencephalography and magnetic resonance imaging results were compatible with HE. HE is diagnosed with differantial diagnosis and exclusion of other reasons. This uncommon disorder is not recognised enough. High titres of serum antithyroid antiboides are always needed for diagnosis. Correct diagnosis requires awareness of wide range of cognitive and clinical presentations of HE.

#### Keywords

Hashimoto's Encephalopathy; Aphasia; Neurological Disorder

DOI: 10.4328/JCAM.3859 Received: 31.08.2015 Accepted: 09.09.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 704-6 Corresponding Author: Şevki Hakan Eren, Acil Tıp AD. Cumhuriyet Üniversitesi Tıp Fakültesi, Sivas, Türkiye.
GSM: +905062379579 E-Mail: shakaneren@hotmail.com

#### Introduction

HE is a rare disease which is sensitive to steroids. Diagnosis is made with exclusion of other reasons of encephalopathy and finding positive results for antithyroid antibodies. This syndrome was first described at 1966 by Brain et al[1]. High levels of serum antithyroid antibodies are essential for diagnosis of HE. Autoimmune encefalopathy associated with HE has variable clinical and psychological symptoms reported at case reports[2]. Thyroid pathologies have widespread prevalance at society. At this case report; we present a male patient with HE and aphasia associated with hypothyroidism who had delayed diagnosis.

#### Case Report

Our patient is a 60-year-old male retired doorman who doesn't have history of diabetes, hypertension, usage of alcohol and tobacco. He had problems of forgetfulness, impairment of attention and concentration disorder for 9 months. He admitted to neurology clinic due to speech complaints continuing for 2 days. Patient's symptoms include forgetting of familiar names and places of his belongings. For last two days, aphasia added to patient's clinical status. Cognitive examination of patient showed attention deficit, impairment of executive functions, loss of visual memory and computation disorder. Myoclonus was present at both upper and lower extremities. Mini Mental Test evaluation was imposibble due to presence of aphasia. Moderate generalised cortical atrophy was found at cranial MR imaging(Fig.1)...

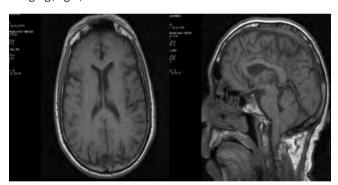


Fig.1. T1- Weighted axial and sagittal cranial MRI showed mild generalized cortical atrophy

Antithyroglobulin antibody level was 600/ml (referance value<34 ml), thyroid function test results were compatible with hypothyroidsm. T3:0,260 ng/ml (2-4,4 ng/ml) T:0,045 ng/ml (0,93-1,70 ng/ml)

Patient's EEG findings at admission were interpreted as generalized slowdown and intermittent rhythmic delta nerve activity . Thyroid ultrasonography showed diffuse heterogeneous and hypoechogen gland.

With these neurological findings and high positivity of anti thyroid antibodies besides the exclusion of another causes of encephalopathy; we have diagnosed the patient with HE and hypothyroidsm. Therapy with 100 mg / day thyroxine and dexamethasone 10 mg / day was started. Patient's symptoms resolved with the treatment of levothyroxin and steroid and he resumed to normal life. Mini mental test score has raised to 29. But his cognitive evaluation showed no development at attention disorder. Aphasia has recovered after two days of steroid

admission. Patient's control EEG results 2 months after his hospital discharge were normal.

#### Discussion

HE is an uncommon clinical syndrome. Diagnosis of this rare disorder requires positive anti TPO level and exclusion of other cases for encephalopathy. Prevalance is reported 2,1/100.000 and male/female ratio for adults is 1:4[3]. Until present approximately 130 cases were reported and most of these cases were female. In contrast to female predominance of these cases our patient was male[4].

Etiology and pathophysiology of HE could not be understood clearly. These patients have increased sensitivity at different otoimmune processes. Antithyroid antibodies and unidentified antibodies which effects brain functions can be the culprit. Possible mechanisms include cerebral vasculitis, toxic effects of TSH and antibodies to central nervous system[5]. Another opinion suggests that thyroid antibodies bind to cerebellar astrocytes and cause pathojenic effects[6]. Two type of presentation for HE has been reported. Focal neurologic symptoms and cognitive dysfunctions can be seen at type with vasculitis. Other type causes progressive and diffuse deterioration resulting confusion, seizures, psychosis, dementia, fast or slow progressing cognitive impairment and somnolence. Myoclonus, tremor, ataxia, focal or generalised seizures, psychiatric symptoms, visual hallucinations can be seen at diffuse type[4,7].

Both of these types can be seen together and these symptoms are potentially reversible[8]. Plenty of diseases like metabolic, toxic, vasculer and neoplastic disorders have similar characteristics with HE; noticing HE requires careful differential diagnosis[4]. HE presents itself with clinical, labaratuary and imaging findings. Clinical symptoms are usually progressive[8]. Tremor, transient aphasia, seizures, gait ataxia, somnolence, myoclonus, neuropsychiatric symptoms and stroke like condition can occur in 1-7 days of HE[4].

Our patient had expressive aphasia. Focal or tonic-clonic seizures were reported two thirds of the cases. Psychosis is present %85 of the patients[9]. Our patient had myoclonus and depression for one month. Elevated antithyroid antibody levels at serum is an important feature of HE. Thyroid antibodies react with brain tissue and these antibodies effect central nervous system. There is a strong correlation between the titres of antibodies and severity of neurologic findings[4,10]. Serum anti TPO and anti Tg levels are elevated at %100 and %73 of the HE cases respectively[4,7].

Autoantibodies against the NH2-terminal of a-enolase (anti-NAE) are reported positive at % 44 of the cases[11]. We could not assess the level of anti-NAE at our patient. Thyroid function test results show variability at the cases of HE. %35 of the cases have subclinical hypothyroidism, %30 of them have normal thyroid functions, %20 of them have evident hypothyroidism and %7 of them have hyperthyroidism[4,9,10]. Our patient had evident hypothyroidism.

Cranial MR imaging can be normal or can show nonspesific findings at white matter. At some studies, %50 of the HE cases had abnormal CT and MR findings such as white matter abnormalities and subcortical or focal cortical abnormalities[7,12]. Our patient had cerebral atrophy at MR imaging, which is a

nonspesific finding. Abnormal electroencephalography (EEG) results and high total protein levels at Cerebrospinal fluid (CSF) are seen at many of the HE cases[13]. Our patient's EEG results at admisson showed generalized slowdown and intermittent rhythmic delta activity. These results were concordant with literature.

At a study for treatment, patients received hormone replacament therapy and steroids. %92 of the patients who had hypothyroidism and HE had recovered[12]. Our patient received both corticosteroid and levothyroxine. Long term prognosis of HE patients varies, there is a high response to treatment but HE can also be progressive or relapsing[4,10]. There are delays at HE diagnosis because HE demands awareness of its manifestations diagnosis. Treatment delays or no treatment at HE causes poor prognosis. More than %25 of the HE patients have permanent cognitive impairment[14]. Our patient's follow up to present day for ten months showed euthyroidism and mild forgetfulness, all of other cognitive functions of patient is normal.

#### Conclusion

HE is an already known disorder. Presentation with mild encephalopathy and aphasia can cause difficulties at diagnosis. A complete and careful neuropsychiatric evaluation is needed. Otoimmune encephalopathy related to hypothyroidism has variable neurologic findings as can be seen at our patient, therefore clinicians should be aware of this.

#### Competing interests

The authors declare that they have no competing interests.

- 1. Brain L, Jellinek EH, Ball K. Hashimoto's disease and encephalopathy. Lancet 1966:2(7462):512-4.
- 2. Archambaud F, Calinat S, Regouby Y. Hashimoto encephalopathy: analysis of four cases. Rev Me'd Interne. 2001;22(7):653-9.
- 3. Chong CS, Leung JL, Wong IH. Presenile dementia: a case of Hashimoto's encephalopathy. East Asian Arch Psychiatry 2011; 21(1): 32-6.
- 4. Chong JY, Rowland LP, Utiger RD. Hashimoto encephalopathy: Syndrome or myth? Arch Neurol 2003;60(2):164-71
- 5. Sawka AM, Fatourechi V, Boeve VH. Rarity of encephalopathy associated with autoimmune thyroiditis: A series from Mayo Clinic from 1950 to1996. Thyroid 2002;12(5):393-8.
- 6. Blanchin S. Coffin C. Viader F. Anti-thyroperoxidase antibodies from patients with Hashimoto's encephalopathy bind to cerebellar astrocytes. J Neuroimmunol. 2007;192(1-2):13-20.
- 7. Marshall GA, Doyle JJ. Long term treatment of Hashimoto's encephalopathy. J Neuropsychiatry Clin Neurosci 2006;18(1):14-20.
- 8. Chen PL, Wang PY, Hsu HY. Reversible electroencephalographic and single photon emission computed tomography abnormalities in Hashimoto's encephalopathy. J Chin Med Assoc. 2005; 68(2):77-81.
- 9. Castillo P, Woodruff B, Caselli R. Steroid responsive encephalopathy associated with autoimmune thyroiditis. Arch Neurol 2006:63(2):197-202.
- 10. Ferracci F, Bertiato G, Moretto G. Hashimoto's encephalopathy: epidemiologic data and pathogenetic considerations. J Neurol Sci 2004, 217(2):165-8
- 11. Kishitani T, Matsunaga A, Yoneda M. The biomarker and treatment in Hashimoto's encephalopahty Nihon Rinsho 2013;71(5):893-7.
- 12. Yuceyar N, Karadeniz M, Copur A. Thyrotoxic autoimmune encephalopathy in a female patient: only partial response to typical immunosuppressant treatment and remission after thyroidectomy. Clin Neurol Neurosurg 2007;109(5):458-62.
- 13. Mocellin R, Lubman DI, Lloyd J. Reversible dementia with psychosis: Hashimoto's encephalopathy. Psychiatry Clin Neurosci 2006;60(6):761-3
- 14. Tzakas P, Sit SW. Progressive impairment of cognition and motor function: Hashimoto encephalopathy. CMAJ 2011; 183(8);495-7.

#### How to cite this article:

Özdemir MT, Atalay E, Tur BK, Eren ŞH, Omurca F, Gürsoy G. Aphasia, Just a Neurological Disorder? J Clin Anal Med 2015;6(suppl 5): 704-6.



# **Delayed Traumatic Intracerebral Hemorrhage: For How Many** Hours Should Patients with Mild Head Trauma be Observed?

İntraserebral Kanama / Intracerebral Hemorrhage

Sevdegul Karadas<sup>1</sup>, Hayriye Gonullu<sup>1</sup>, Ismail Gulsen<sup>2</sup>, Huseyin Baltacıoglu<sup>1</sup> Department of Emergency Medicine, <sup>2</sup>Department of Neurosurgery, Yuzuncu Yil University, School of Medicine, Van, Turkey

Gecikmiş travmatik intraserebral hematom (GTİH) kafa travmasının nadir görülen bir komplikasyondur. GTİH'un oluşum mekanizması tam olarak bilinmemektedir. Biz burada hafif kafa travmalı bir olguda gelişen GTİH olgusu rapor ettik. 25 yaşındaki erkek hasta futbol maçı yaparken düşmüştü. Başvuru anında baş ağrısı ve baş dönmesi vardı. Acil serviste yaklaşık 12 saat boyunca gözlem altında tutuldu. Kafa travmasından 26 saat, bulantı, kusma ve baş ağrısı şikayetleri ile tekrar hastanemize başvurdu. Kontrol beyin bilgisayarlı tomografi çekildi ve frontal bölgede travmatik intraserebral hematom tespit edildi. Sonuç olarak GTİH fatal olabilir. Yakın gözlem ve tekrarlayan BBT komplikasyonları ve mortaliteyi azaltabilir.

#### Anahtar Kelimeler

İntraserebral Kanama; Kafa Travması; Gözlem

#### Abstract

Delayed traumatic intracerebral hematoma is a rare complication of head injury. The etiopatogenesis of DTIH is not precisely known. We herein report a case of delayed traumatic intracerebral hematoma, with mild HT. This 25-year-old male fell down while playing in a footbal match. He had headache and vertigo. He was kept under observation for about 12 hours at the emergency department. At the 26th hour after the HT incident, he presented to our hospital again with the complaints of nausea, vomiting and headache. A control brain computed tomography was performed and a traumatic intracerebral hematoma was determined in the frontal region. As a result, DTIH may be fatal. Close observation and repeat CBT scanning may reduce complications and mortality.

Intracerebral Hemorrhage; Head Trauma; Observation

DOI: 10.4328/JCAM.3841 Corresponding Author: Sevdegul Karadas, Department of Emergency Medicine, Yuzuncu Yil University, Faculty of Medicine, Van, Turkey. T.: +90 4322150470/6765 F.: +90 4322167519 E-Mail: sevdegulkaradas@gmail.com

#### Introduction

Head trauma (HT) is one of the most important public health problems. The incidence of minor HT has been reported to range from 130 to 200 cases per 100,000 people per year, although the estimates have been as high as 500 cases per 100,000 people per year [1]. Traumatic intracerebellar hematoma accounts for 0.6-0.82 % of all head traumas [2]. Patients with HT are evaluated in different groups according to their Glasgow Coma Scale (GCS) score. GCS of 13-15 indicates mild HT, 9-12 indicates moderate HT and 3-8, severe HT [3].

Delayed traumatic intracerebral hemorrhage (DTIH) is rare and is shown by brain computed tomography (BCT), where the hematoma develops several hours after the trauma [2]. Its condition and etiopatogenesis are still controversial and there is no standardization in terms of incidence, classification and treatment [2,4]. The first case of DTIH was described in 1891 by Bollinger. The author said that areas of softening developed in the cerebrum and medulla after head trauma. In later years, another author emphasized that vascular spasm acted a role in the etiology of DTIH [4].

In patients with DTIH, early diagnosis and treatment are very important to decrease mortality and morbidity. Therefore, a control BCT should be performed 4-8 hours after trauma in patients with severe head trauma, coagulation disorders, cranial fracture and patients over the age of fifty [4]. Observation for at least 6-24 hours in patients with minor HT can be suggested. Furthermore, a control BCT should be obtained before discharging these patients [5]. Here, we report a case of delayed traumatic intracerebral hematoma, with mild HT.

#### Case Report

A 25-year-old male fell down while playing in a footbal match. He had headache and vertigo. Upon admission to the hospital, his arterial blood pressure was 130/80 mm Hg, his respiratory rate was 20/min, temperature was 36°C and his O2 saturation level was 98%. His heart rate was 82/min during auscultation, and the other system examinations revealed normal results. He had no history of chronic illness, and no drug or alcohol use. His Glasgow Coma Scale (GCS) score was 15 (E-4, V-5, M-6). His pupils were isochoric and the light reflex was bilaterally positive during his physical examination. No focal finding, no evidence of meningeal irritation or pathologic reflex was noted. On admission, an initial BCT scan was performed revealing normal findings (Figure 1). He was kept under observation for about 12 hours at the emergency department and discharged from the hospital with normal neurological examination signs, normal signs of second BCT (Figure 2) and without complaints. He had no history of abnormally high systemic blood pressure occurring during the observation period. Clinically, no attacks of vertigo or nausea and focal neurological sign occurred. However, at the 26th hour after the HT incident, he presented to our hospital again with the complaints of nausea, vomiting and headache. GCS was 15. A control BCT was performed and a traumatic intracerebral hematoma was determined in the frontal region (Figure 3). He was transferred to the neurosurgery clinic and he was discharged from the hospital without any sequela on the seventh day of his hospital stay.



Figure 1. An initial BCT scan normal findings



Figure 2. Normal neurological examination signs, normal signs of second BCT ( About 12 hours at the emergency department)

#### Discussion

DTIH is a rare complication of head injury [2]. The etiopatogenesis of DTIH is not precisely known. However, the possible pathogenic mechanisms may include impairment of autoregulation, necrosis, structural abnormalities of the vessel walls after injury, traumatic aneurysm, metabolic changes at the cellular level, vasospasm, venous congestion as a result of increased intracranial pressure and coagulopathy at the region of trauma [4].

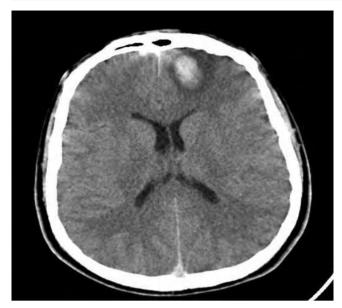


Figure 3. A traumatic intracerebral hematoma was determined in the frontal region

BCT and MRI (Magnetic resonance imaging) is useful in diagnosing intracerebral hematomas. BCT has been particularly helpful at the emergency department in increasing the diagnosed patients population [4,6]. Fukamachi et al. reported that DTIH developed within 48 hours in 50 % of patients in whom CBT was normal or when there was a minimal hyperdense lesion within the first 6 hours [6]. Nagata et al. said that DTIH occured in 54.5 % of patients who had normally appearing areas on the initial BCT [2].

Kaplan et al. stated that DTIH is commonly demonstrated within the first post-traumatic 10 days, particularly in the first 3 days [7]. Nagata et al. said that the time of occurrence of DTIH ranged from 4 hours to 4 days, with a mean of 31.5 hours [2]. In the literature, it has been stated that DTIH usually occurs within 72 hours after HT [4]. In a pediatric patient group with 397 patients, Hamilton et al. reported the rate of DTIH as % 4.3 [1]. However, Nagata et al. [2] reported an average age of 25.5 and reported no gender predominance. In our study, the was 28 years of age and ICH was established 26 hours after the HT event.

Nagata et al. [2] stated that DTIH was usually found in the posterior fossa and that its prognosis was known as unfavorable. However, in another study, it reported that the most common locations for DTIH were the frontal and the temporal lobes [4]. Kaplan et al. [7] said that DTIH was located in the occipitoparietal region by a countercoup mechanism. In our case, the intracerebral hemorrhage was located in the frontal region and he was discharged on the seventh day of hospitalization. Fortunately, our patient completely recovered.

In a study, While the clinical appearance is initially stable in these patients, a suddenly decreased GCS, increased focal neurological sign or focal seizures can ocur [4]. In Kutlay's study, neurological examination was normal in 50% of cases with DTIH [4]. In our study, the patient's clinical situation was stable and his neurological examination was normal. We performed CBT initially and 12 hours after admission. He had normal CBT findings. However, he presented again to our emergency department 26 hours after the HT event and an intracerebral hematoma was determined.

The use of emergency CBT is still controversial in patients with mild HT [8]. In previous studies, it has been reported that the rates of detection of abnormal signs on the CBT varied in patients with mild HT [8]. Furthermore, mortality and neurosurgical intervention are rare in mild HT. Nevertheless, Kreitzer et al. reported that patients with mild HT demonstrated a low rate of adverse outcomes when observed for 6-24 hours [5].

Our case had a favorable outcome without surgery, but DTIH may be fatal. Close observation and repeat CBT scanning render more favorable results. Therefore, patients with mild HT should undergo close observation and they should be given advice regarding HT after discharge. Furthermore, the observation period may be extended and an outpatient control should be proposed within three days for patients with mild HT.

Conflict of interest; The authors further declare that we have no financial arrangement as product in the case report, sources of funding, institutional affiliations, and any possible financial or personal conflicts of interest.

#### Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Hamilton M, Mrazick M, Johnson D. Incidence of Delayed Intracranial Hemorrhage in Children After Uncomplicated Minor Head Injuries. Pediatrics 2010; 1(126) · e33
- 2. Nagata K, Ishikawa T, Ishikawa T, Shigeno T, Kawahara N, Asano T, et al. Delayed traumatic intracerebellar hematoma: correlation between the location of the hematoma and the pre-existing cerebellar contusion: case report. Neurol Med Chir 1991;31(12):792-6
- 3. Cete Y, Pekdemir M, Oktay C, Eray O, Bozan H, Ersoy FF. The role of computed tomography for minor head injury. Ulus Travma Dergisi 2001;7(3):189-94.
- 4. Kutlay M, Kıbıcı K, Demircan N. Early diagnosis, Follow up and Treatment of Delayed Traumatic intracerebral haematomas. Turkish Journal of Trauma- Emergency Surgery 1998;4(1):33-8.
- 5. Kreitzer N, Lyons MS, Hart K, Lindsell CJ, Chung S, Yick A, et al. Repeat neuroimaging of mild traumatic brain-injured patients with acute traumatic intracranial hemorrhage: clinical outcomes and radiographic features. Acad Emerg Med 2014 ;21(10):1083-91
- 6. Fukamachi A, Nagaseki Y, Kohno K, Wakao T. The incidence and developmental process of delayed traumatic intracerebral haematomas. Acta Neurochir (Wien) 1985;74(1-2):35-9.
- 7. Kaplan M., Ozveren MF, Topsakal C., Erol FS, Akdemir I, Asymptomatic interval in delayed traumatic intracerebral hemorrhage: report of two cases. Clin Neurol Neurosurg 2003;105(3):153-5.
- 8. Aygün D, Güven H, İncesu L, Şahin H, Doğanay Z, Altıntop L. The incidence of pathologic findings of the cranial computed tomography in patients with minor head trauma and its correlation with age and clinical features. Ulus Travma Derg 2003;9(2):129-33.

#### How to cite this article:

Karadas S, Gonullu H, Gulsen I, Baltacioglu H. Delayed Traumatic Intracerebral Hemorrhage: For How Many Hours Should Patients with Mild Head Trauma be Observed? J Clin Anal Med 2015;6(suppl 5): 707-9.

# CAM Can and Ca

## Pain in the Right Lower Quadrant Without Appendix

## Appendiks Olmadan Sağ Alt Kadranda Ağr

Appendiks Olmadan Sağ Alt Kadranda Ağrı / Right Lower QuadrantPain Without Appendix

İhsan Yıldız, Yavuz Savas Koca, Koray Okur, Bekir Sarıcık Department of Genaral Surgery, Suleyman Demirel University, School of Medicine, Isparta, Turkey

#### Ozet

Apendiksin agenezisi birçok deneyimli cerrah tarafından bile nadir görülen klinik bir durumdur. Bu duruma akut apandisit tanısıyla ameliyata alınan 100 000 olguda 1 olarak rastlanılmaktadır. Daha önce herhangi bir operasyon geçirmemiş 20 yaşındaki erkek hasta akut apandisit semptomlarıyla acil servise kabul edilip ameliyata alındı. Ancak yapılan eksplorasyonda apendiks bulunamadı. Akut apandisit semptomlarıyla gelen olgudan öncelikle dikkatli bir anamnez alınmalı, özellikle geçirilmiş operasyonlar bakımından iyi sorgulanmalı ve muayene edilmelidir. Apendiks agenezisi tanısına karar vermeden önce ileo-çekal bölge ve çıkan kolon mobilize edilerek kapsamlı ve titiz bir ekplorasyon yapılmalıdır. Ayrıca bütün bu işlemler yapılırken hassas olan ileo-çekal bölge travmatize edilmemelidir. Akut apandisit semptomları olan olgularda appendiks agenezisi olabileceği akılda bulundurulmalıdır.

#### Anahtar Kelimeler

Apendiks Yokluğu; Apendiks Agenezisi

#### Abstract

Appendix agenesis is rarely seen, even for many experienced surgeons. The incidence of appendix agenesis has been reported as 1 per 100,000 cases admitted for surgery with a diagnosis of acute appendicitis. A 20-year old male with no history of surgery presented to the emergency department with the symptoms of acute appendicitis and was admitted for surgery. No appendix was found in the surgical exploration. Cases presenting with the symptoms of acute appendicitis should have a detailed anamnesis taken with careful questioning, particularly related to previous surgery and a thorough physical examination should be performed. Before establishing the diagnosis of appendix agenesis, a comprehensive and careful exploration should be conducted after mobilizing the ileo-cecal region and ascending-colon. In addition, care should be taken to avoid injuring the ileo-cecal region, which is highly sensitive. Appendix agenesis should be kept in mind in cases presenting with the symptoms of acute appendicitis.

#### Keywords

Appendix Absence; Appendix Agenesis

DOI: 10.4328/JCAM.3874 Received: 08.09.2015 Accepted: 29.09.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 710-1 Corresponding Author: İhsan Yıldız, Genel Cerrahi Anabilim Dalı, SDU Tip Fakultesi, 32100 Isparta, Türkiye.
GSM: +905055615647 F.: +90 2462234284 E-Mail: drihsanyildiz@gmail.com

#### Introduction

Congenital absence of the appendix is defined as the agenesis or atresia of appendix. Appendix agenesis was first described by Morgagni in 1718 [1]. The incidence of appendix agenesis in laparotomies applied with initial diagnosis of acute appendicitis has been reported as 1/100,000 [1, 2]. Diagnosis of appendix agenesis is made by laparotomy or laparoscopy [1-3].

In this report, we present a case who was initially diagnosed with acute appendicitis symptoms by diagnostic laparoscopy and later diagnosed with vermiform appendix agenesis during surgical exploration. This uncommon clinical event is presented in light of the literature.

#### Case Report

A 20-year old male with no history of previous surgery presented to the emergency department with a two-day history of nausea, vomiting, lack of appetite and abdominal pain which had started in the in the right lower quadrant.

The patient had no history of previous surgery and no sign of appendicitis such as perforated appendix. On auscultation, the intestinal sounds were normoactive. In the abdominal examination, there was sensitivity and muscular defense on palpation in the right lower quadrant. No clinical sign was found in the rectal examination and the other system examinations were normal. Laboratory findings were as follows: leukocytes; 11,400/ml (4000-8000) and other biochemistry results were normal. No appendix was observed on ultrasound. The findings initially suggested the presence of acute appendicitis. In the diagnostic laparoscopy, since the appendix was not visualized in the location of the cecum, the cecum was mobilized and the exploration was extended from the ileo-cecal region to ascending-colon; however, no appendix was found. The patient was discharged on the second day after operation since the complaints recovered and the laboratory parameters returned to normal. The patient has been followed up for a year and no problem has been detected so far.

#### Discussion

Appendix agenesis is a rarely seen clinical event which was first described by Morgagni in 1718. Although the appendix agenesis is likely to be diagnosed during abdominal surgery, the definitive diagnosis is made by laparoscopy or laparotomy [1-3]. In our study, the patient had no history of previous surgery. The diagnosis of vermiform appendix agenesis was established during diagnostic laparoscopy. In such situations, the presence of similar previous symptoms should be investigated [4-5]. If laparotomy is performed with the suspicion of acute appendicitis, all the possibilities that mimic these symptoms should be investigated. Moreover, the patient should be questioned as to whether or not these or similar symptoms had previously occurred. Most common of these symptoms include terminal ileitis, ectopic pregnancy rupture, Meckel's diverticulum, mesenteric lymphadenitis (more often in children), salpingo-oophoritis, and ovarian cyst rupture. In some cases, the appendix may be resorbed by autoamputation following perforated appendicitis [4]. Urinary infections and the stones at the lower ureter may also exhibit similar symptoms. However, none of these symptoms had been experienced by our patient. The absence of ap-

pendix may be in the form of congenital agenesis or atresia of appendix, or rudimentary appendix [6-8]. Shand et al reported that the use of thalidomide during pregnancy caused appendix agenesis during pregnancy (in utero) [6]. In our study, the patient had no history of in-utero usage of thalidomide. In some studies, appendix agenesis has been reported with intestinal malformations [7-8]. In our patient, no intestinal malformation was observed. To avoid incorrect diagnosis, history of previous surgeries and appendectomy should be carefully investigated [2]. In our patient, no history of surgery or intra-abdominal infection that could cause autoamputation was detected.

When the appendix cannot be seen easily, the cecum should be fully mobilized, the site of the convergence of the three teniacoli and ascending colon should be explored, and the ileo-cecal region should be carefully examined [1-4]. In our patient, although the exploration was extended from the convergence of the three tenia coli to ascending colon, no appendix was found. As above mentioned, there may be several reasons for the pain in the right lower quadrant. When there is no positive sign, a stone at the lower ureter is the most probable cause. In our patient, no positive sign that could cause the pain in the right lower quadrant was found, except for the stone at the lower ureter which had fallen spontaneously. We considered that this situation was the reason for the spontaneous recovery of the symptoms

The aim of this report was to emphasize the clinical importance of appendix agenesis, rather than the reasons causing this clinical event. Accordingly, we believe that the presentation of this rare case will be beneficial for the early diagnosis of patients with appendix agenesis who present with a pain in the right lower quadrant.

## Conclusion

Vermiform appendix agenesis is a rare occurrence. Medical history of the patient is of prime importance since resorption might have occurred in perforated appendicitis or the appendix might have been removed in a previous laparotomy.

## Competing interests

The authors declare that they have no competing interests.

#### References

- 1. Vincent MV, Doyle A, Bernstein S. Absence of the appendix discovered during childhood. Springer plus 2014;3:522.
- 2. Chevre F, Gillet M, Vuilleumier H. Agenesis of the vermiform appendix. Surg Laparosc Endosc Percutan Tech 2000;10(2):110-2.
- 3. Yaylak F, Algin MC, Gungor Y. Atresia of the appendix vermiformis: a rare case of developmental abnormality. World J Gastroenterol 2013;19(1):122-4.
- 4. Lima M, Antonellini C, Aquino A. Agenesis of the appendix vermiformis. Pediatr Med Chir 2003:25(5):370-2.
- 5. Anyanwu SN. Agenesis of the appendix:casereport. West Afr I Med 1994:13(1):66. 6. Shand IE. Bremner DN. Agenesis of the vermiform appendix in a thalidomide child. Br J Surg 1977;64(3):203-4.
- 7. Cserni T, Magyar A, Németh T. Atresia of the ileocecal junction with agenesis of the ileocecal valve and vermiform appendix: report of a case. Surg Today 2006;36(12):1126-8
- 8. Yokose Y, Maruyama H, TsutsumiM. Ilealatresia and absence of appendix. Acta Pathol Jpn 1986;36(9):1403-10.

#### How to cite this article:

Yıldız İ, Koca Y.S, Okur K, Sarıcık B. Pain in the Right Lower Quadrant Without Appendix. J Clin Anal Med 2015;6(suppl 5): 710-1.



# Glenoid Dysplasia in the Recurrent Shoulder Dislocation: A Case Report

# Tekrarlayan Omuz Çıkıklarında Glenoid Displazi: Bir Olgu Sunumı

Omuz Çıkıklarında Glenoid Displazi / Glenoid Dysplasia in Shoulder Dislocation

Hamza Sucuoğlu<sup>1</sup>, Kubilay Ersin Türkmen<sup>2</sup>, İslam Çalışkan<sup>3</sup>, Nurettin İrem Örnek<sup>4</sup>

<sup>1</sup>Fiziksel Tıp ve Rehabilitasyon Kliniği, Özel Bağcılar Aktif Tıp Merkezi, İstanbul,

<sup>2</sup>Ortopedi ve Travmatoloji Kliniği, Özel Mardin Park Hastanesi, Mardin,

<sup>3</sup>Ortopedi ve Travmatoloji Kliniği, Silopi Devlet Hastanesi, Şırnak,

<sup>4</sup>Fiziksel Tıp ve Rehabilitasyon Kliniği, Özel Safir Tıp Merkezi, İstanbul, Türkiye

Bu olgu sunumu 22-26 Nisan 2015 tarihinde Antalya-Türkiye'de gerçekleştirilen 25. Ulusal Fiziksel Tıp ve Rehabilitasyon kongresinde bildiri olarak sunulmuştur.

#### Özet

Glenoid displazi sıklıkla gözardı edilen, skapulanın nadir bir gelişimsel anomalisidir. Birçok hastada asemptomatik olup, çekilen akciğer grafisinde rastlantısal olarak teşhis edilmesine rağmen omuz ekleminde insitabilite oluşturması nedeniyle de tarif edilmiştir. Bizim genç erkek hastamızda son 5 yıldır, yılda 4-5 kez tekrar eden omuz çıkıkları oluyormuş. Her defasında acil poliklinikte redüksiyon yapılıp, omuz askısı ve istirahatle taburcu ediliyormuş. Son olarak tekrar gerçekleşen omuz çıkığı ile tarafımıza başvurduğunda ise, çekilen direkt grafisinde bilateral glenoid displazi tanısı konuldu. Bu olgumuzdaki gibi tekrarlı omuz çıkıkları olan
hastalarda akla gelmesi gereken glenoid displazi tanısını, klinik ve radyolojik bulguları ile birlikte sunuyoruz.

#### Anahtar Kelimeler

Glenoid displazi, Omuz çıkığı, Radyografi, Rehabilitasyon

#### Abstract

Glenoid dysplasia, which is often ignored, is a rare developmental anomaly. In most cases the patients remain asymptomatic. Although glenoid dysplasia has been diagnosed by incidentally on chest radiograph, also it has been described because of the developing instability of shoulder joint. Our young male patient who has recurrent dislocation of the shoulder-joint 4-5 times a year for last 5 years, dislocation has been reduced in the emergency department and he has been discharged from hospital with shoulder strap and rest. Finally when he admitted to our outpatient clinic with recurrent shoulder dislocation, the diagnosis of glenoid dysplasia is revealed by X-ray examination. We present the diagnose of glenoid dysplasia with clinical and radiological findings which should be kept in mind in patients with recurrent dislocations of shoulder as in our case.

#### Keywords

Glenoid dysplasia, Shoulder dislocation, Radiography, Rehabilitation

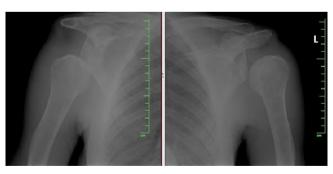
DOI: 10.4328/JCAM.3878 Received: 10.09.2015 Accepted: 30.09.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 712-4 Corresponding Author: Hamza Sucuoğlu, Fiziksel Tıp ve Rehabilitasyon Kliniği, Özel Bağcılar Aktif Tıp Merkezi, Fatih Mah. İlke Sok. No:1, Bağcılar, İstanbul, Türkiye. GSM: +905056297688 F.: +90 2126526325 E-Mail: hamzasucuoglu@mynet.com.tr

#### Giris

Glenoid displazi, skapula glenoid kıkırdağın alt üçte ikisinin eksik kemikleşmesi ile karakterize nadir bir durumu ifade eder [1,2]. Sıklıkla göz ardı edilen bu durum birçok hastada asemptomatik olup, çekilen akciğer grafisinde rastlantısal olarak teşhis edilmesine rağmen, omuz ekleminde insitabilite oluşturması nedeniyle de tarif edilmiştir [3]. Wirth ve ark. [3] bu hastaları üç grup olarak tarif etmişlerdir. Bir; omuz insitabilitesi olmadan bilateral glenoid hipoplazisi olanlar, iki; insitabilite ile bilateral glenoid hipoplazisi olanlar ve üçüncüsü; tek taraflı glenoid hipoplazisi ve humerus başının ciddi deformitesi olanlar. Bizim hastamızda, ikinci grupta; insitabilite ile bilateral glenoid hipoplazisi ve buna bağlı tekrarlı omuz çıkıkları olan bir hastaydı. Biz de, olgumuzdaki gibi tekrarlı omuz çıkıkları olan hastalarda glenoid displazinin akla gelmesi gerektiğini, bu hastalığın klinik ve radyolojik bulguları ile birlikte tanısını ve tedavisini sunuyoruz.

#### Olgu Sunumu

Her iki omuz ağrısı ve hareket kısıtlılığı şikayeti ile başvuran 33 yaşında erkek hastanın ağrıları işte çalışırken gelişen omuz çıkıkları sonrası meydana geliyormuş. Her iki omuzda da gelişebilen çıkıklar, son 5 yıldır, yılda 4-5 kez oluyormuş. Her seferinde acil polikliniğe başvuran hastanın omuz çıkığı düzeltiliyor, omuz askısı ve istirahat tedavisi ile 1-2 hafta içerisinde tekrar işine geri dönüyormuş. Son olarak bir gün önce sağ omuzunda tekrar çıkık olmuş ve acil servise başvuran hasta tarafımıza yönlendirilmiş. Hastanın muayenesinde sağ omuz eklem hareketleri fleksiyon 140, abduksiyon 130, iç ve dış rotasyon 70 derecede kısıtlı ve şiddetli ağrılıydı. Sol omuz hareketleri ise; abduksiyon ve fleksiyon 160 derecede, iç ve dış rotasyon 80 derecede kısıtlı ve hafif ağrılıydı. Sol omuzda Neer ve Hawkins sıkışma testleri pozitif saptandı. Omuz stabilite muayenesinde bilateral omuz aprehansion (endişe) testi pozitif bulundu. Genel eklem laksitesi için yapılan değerlendirmede hiçbir anormal bulgu yoktu. Kas kuvveti, derin tendon refleksleri ve duyu muayenesi normaldi. Çekilen antero-posterior omuz grafisinde sağ humerus başının glenoid inferiorunda olduğu ve her iki skapula boynunun kısa ve glenoidin hipoplazik olduğu görüldü (Resim 1). Bu



Resim 1. Bilateral antero-posterior omuz grafisi: Her iki skapula boynunda kısalık, glenoid fossada sığlık, korakoidde uzama ve sağda belirgin humerus başı glenoid inferiorunda lokalize görülmektedir

bulgular ışığında, omuz eklemleri insitabil olan hastaya bilateral glenoid displazi tanısı konuldu. Hastanın sağ omuz eklemi kapalı redükte edildi. Redüksiyon sonrası nörovasküler muayenesi doğaldı. Hasta konservatif tedaviye alınarak; sağ omuzu nötral pozisyonda ve hafif abdüksiyonda tutan yastık destekli Velpeau bandajı, anti-inflamatuar ilaçlar, sıcak uygulama ve yaşam tarzı değişiklikleri verildi. Beraberinde hasta, 6 hafta süren (30

seans) fizik tedavi ve rehabilitasyon (FTR) programına alındı. Bu programda; her iki omuz için ultrason (1MHz frekans, 1.5 W/ cm2 yoğunluk, 10 dakika /seans), transkütanöz elektrik sinir stimülasvonu (TENS ) (konvansivonel mod. 100Hz frekans. 100us uyarı süresi, 20 dakika/seans), supraspinatus, deltoid ve infraspinatus kaslarına elektrostimülasyon (kuvvetlendirme programı, 20-50Hz frekans, 0-100 mA amplitüd, optimal kontraksiyona göre uyarı süresi, 20 dakika/seans) uygulamaları yapıldı. Ek olarak, bilateral glenohumeral ve skapulatorasik eklem stabilizasyon ve kuvvetlendirme (izometrik ve izotonik) egzersizleri uygulandı. FTR programı sonrası omuz ağrıları ve hareket açıklığı tama yakın düzelen hasta, ev egzersiz programı ile taburcu edildi. Ayrıca hasta, glenohumeral eklem kapsülü ve varsa labral lezyonların değerlendirilmesi için her iki omuz manyetik rezonans (MR) artrografi çekilmesi ve cerrahi açıdan takibinin yapılması için bir üst merkeze yönlendirildi.

#### Tartışma

Glenoid displazinin etiyolojisi tam olarak anlaşılamamıştır. Patogenezinde, alt glenoid kıkırdak kemikleşmesinde bir aksama olduğu görülmektedir [2]. Doğumda skapulanın büyük bir kısmı kemikleşmiş olmasına rağmen, skapulanın alt köşesi, akromiyon, korakoid proses ve glenoidde doğum sonrası yaklaşık on-on beş yıla kadar kıkırdak doku varlığını sürdürmektedir. Glenoid fossa bir proksimal ve distal kemikleşme merkezi ile gelişir. Bu ossifikasyon merkezlerinin biri veya her ikisinin birlikte sapma göstermesi glenoid displazi gelişiminde rol oynamaktadır. Olguların az bir kısmı ailesel olmasına rağmen, bizim olgumuzdaki gibi çoğunda aile öyküsü yoktur [1]. Glenoid displazi önce nadir görülen bir durum olarak kabul edilmesine rağmen, son yıllarda düşünülenden daha yaygın olabileceği varsayılmaya başlanmıştır. Bunun nedeni olarak ise, sıklıkla asemptomatik olması nedeniyle insidansının geçmişte göz ardı edilmesi olarak açıklanmaktadır [4]. Ayrıca Keith ve ark. [4] yaptıkları çalışmada; 2 aylık bir sürede 98 hastanın MR artrografilerini retrospektif olarak incelediklerinde, %14.3'ünde orta veya şiddetli glenoid displazi saptamışlardır. Edelson [5] ise, 11.000'in üzerinde kadavrayı incelediğinde kuzey Çin ve Alaska Eskimo toplumlarında görülen glenoid displazi insidansının % 19 ile % 35 arasında değiştiğini bildirmiştir.

Glenoid displazi birçok hastada asemptomatik olup, çekilen akciğer grafisinde rastlantısal olarak teşhis edilir. Bizim olgumuzda olduğu gibi, genellikle bilateral ve simetrik olup, bazen tek taraflı olabilmektedir [1]. Literatür bilgileri, olguların muhtemelen hayatın ikinci veya üçüncü on yılında semptomatik olacağını bildirmektedir [1]. Glenoid displazisi olan hastalarda ağrı, hareket kısıtlılığı ve omuz insitabilitesi değişken sıklıklarda görülmektedir. Literatür gözden geçirildiğinde, hastaların yaklaşık % 21'inde omuzda ağrı, %43'ünde omuz hareketlerinde kısıtlılık ve %2'sinde semptomatik insitabilite vardır [1]. İkinci on yıldan sonra semptomatik hale gelen hastamızda; omuz ağrısı, tekrarlı omuz çıkıkları (insitabilite) ve hareket kısıtlılıkları vardı.

Glenoid displazide görülen radyografik bulgular, literatürde tarif edilmiştir. Bunlar; skapula boynunda kısalık, glenoidde sığlaşma ve korakoid ile klavikulada uzama ve şekil değişikliklerini içermektedir [6]. Glenoid alt köşesinde çentikleşme ve düzensizlik tipik olarak görünür. Ayrıca glenohumeral eklem alt köşesinin büyümesi de, muhtemelen glenoid labrum hiperplazisine ikincil olarak tarif edilmiştir [7]. Bizim olgumuzun radyografisinde de bilateral tipik skapula boynundaki kısalık ve glenoid hipoplazi görülmekteydi.

Glenoid displazide MR bulguları ise, glenoid fossa artiküler kıkırdağın kalınlaşması ve posterior glenoid labrumun genişlemesidir. MR artrografide ise anormal inferior glenoid labrumda fissür görülebilmektedir [7].

Glenoid displazi tedavisi tartışmalı olmakla birlikte, çoğu yazar konservatif yaklaşımları savunmaktadır [3]. Wirth ve ark. [3] tedavide FTR programlarının semptomları gidermede etkili olduğunu bildirirken, Smith ve ark. [8] ise; 12 hastalık olgu serilerinde, genç hastalarda FTR'nin daha başarılı olduğunu fakat yaşlılarda ameliyata ihtiyaç olabileceğini bildirmişlerdir. Bizim olgumuzda ise, FTR programına hastanın uyumunun ve katılımın yüksek olması ve genç yaşta olması nedeniyle tedavinin başarılı olduğu düşünülmektedir. Glenoid diplazi hastalarının cerrahi tedavisinde ise, yaş, semptomların süresi, osteoartrit varlığı ve insitabilite gibi faktörlere bağlı olarak cerrahi yaklaşımlar farklılık göstermektedir [8]. Eğer insitabilite ile birlikte glenoid displazi erken fark edilirse, bir glenoid osteotomi glenoidin normal versiyonunu yeniden sağlayabilir [7]. Ayrıca, bu bozuklukla birlikte glenohumeral artriti olan yaşlı hastalarda ise, total omuz replasmanı yapılabileceği bildirilmektedir [7]. Olgumuz konservatif yaklaşımla tedavi edilmesine rağmen, cerrahi açıdan daha iyi bir değerlendirme yapılabilmesi ve takibi için üst bir merkeze yönlendirilmiştir.

Sonuç olarak; glenoid displazi sıklıkla gözden kaçabilen, radyolojik bulgularının bilinmesi ile farkında olunabilecek gelişimsel bir omuz anomalisidir. Omuz ağrısı ve hareket kısıtlılığı yanında tekrarlı omuz çıkıklarında da akla gelmesi gerekmektedir. Bu hastalarda tipik radyografi ve MR artrografi bulguları ile tanı konulabilmektedir. Glenoid displazi tedavisinde ise FTR uygulamaları etkili olmakla birlikte cerrahi girişimlere de ihtiyaç duyulabilmektedir.

#### Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı beyan edilmemiştir.

## Kavnaklar

- 1. Survawanshi A. Mittal A. Dongre S. Kashyap N. Unilateral glenoid hypoplasia: A case report and review of the literature. Case Rep Orthop 2011; DOI: 10.1155/2011/412721.
- 2. Currarino G, Sheffield E, Twickler D. Congenital glenoid dysplasia. Pediatr Radiol 1998;28(1):30-7.
- 3. Wirth MA, Lyons FR, Rockwood CA. Hypoplasia of the glenoid. A review of sixteen patients. J Bone Joint Surg Am 1993;75(8):1175-84.
- 4. Harper KW, Helms CA, Haystead CM, Higgins LD. Glenoid dysplasia: incidence and association with posterior labral tears as evaluated on MRI. AJR Am J Roentgenol 2005;184(3):984-8.
- 5. Edelson JG. Localized glenoid hypoplasia. Clin Orthop Relat Res 1995;(321):189-
- 6. Kavanagh EC, Zoga AC, Abboud JA, Kourlouris G, Morrison WB. Glenoid dysplasia: radiographic, direct MR arthrographic and arthroscopic appearances. Radiology Case Reports 2006; DOI: 10.2484/rcr.v1i2.20.
- 7. Trout TE, Resnick D. Glenoid hypoplasia and its relationship to instability. Skeletal Radiol 1996;25:37-40.
- 8. Smith SP, Bunker TD. Primary glenoid dysplasia. A review of 12 patients. J Bone Joint Surg Br 2001;83(6):868-72.

#### How to cite this article:

Sucuoğlu H, Türkmen K.E, Çalışkan İ, Örnek N.İ. Glenoid Dysplasia in the Recurrent Shoulder Dislocation: A Case Report. J Clin Anal Med 2015;6(suppl 5): 712-4.



# Non-Type B Haemophilus Influenzae Meningitis: A Case Report

Tiplendirilemeyen Haemophilus İnfluenzae / Non-Type B Haemophilus İnfluenzae

Fatma Deniz Aygün<sup>1</sup>, Ada Bulut Sinoplu<sup>2</sup>, Fatma Köksal Çakırlar<sup>3</sup>, Haluk Çokuğraş<sup>1</sup>, Yıldız Camcıoğlu<sup>1</sup> Cocuk Sağlığı ve Hastalıkları AD, Enfeksiyon Hastalıkları, Klinik İmmünoloji ve Allerji BD, İstanbul Üniversitesi, Cerrahpaşa Tıp Fakültesi, <sup>2</sup>Çocuk Sağlığı ve Hastalıkları AD, İstanbul Üniversitesi, Cerrahpaşa Tıp Fakültesi, <sup>3</sup>Tıbbi Mikrobiyoloji AD, İstanbul Üniversitesi, Cerrahpaşa Tıp Fakültesi, İstanbul, Türkiye

Makalemiz 22-26 Ekim 2014 tarihinde Antalya-Belek Kaya Palazzo Otel'de yapılmış olan 58.Türkiye Milli Pediyatri Kongresi'nde poster olarak sunulmuştur.

Haemophilus influenzae çocukluk çağında önde gelen menenjit etkenlerindendir. Beş yaş altında invaziv enfeksiyonlara neden olur ve Haemophilus influenzae Tip b (Hib) serotipi vakaların büyük çoğunluğundan sorumludur. Konjuge Hib aşılarının rutin aşı takvimine girmesinden sonra invaziv enfeksiyonların sayısı belirgin olarak azalsa da aşılamaya karşın halen invaziv hastalık bildirilmekte ve bu olgular arasında kapsülsüz (tiplendirilemeyen) serotiplere rastlanmaktadır. Ancak tiplendirilemeyen serotiplerin neden olduğu invaziv hastalık sıklığında artış ile ilgili henüz kesin bir veri yoktur. Her yaş grubunda akut bakteriyel menenjit etkenleri arasında tiplendirilemeyen Haemophilus influenzae serotipine rastlanmaktadır. Bu yazıda, Hib aşısı yapılmış bir çocukta tiplendirilemeyen H.influenzae enfeksiyonuna bağlı bakteriyel menenjit olgusu etkene dikkat çekmek amacıyla sunulmuştur.

#### Anahtar Kelimeler

Tiplendirilemeyen Haemophilus İnfluenzae; Menenjit; Çocuk

#### Abstract

Haemophilus influenza is one of the most common cause of bacterial meningitis in children. H.influenzae, especially type b (Hib) serotype causes invasive infections in children under five years of age. The widespread use of Hib conjugate vaccines has led to a dramatic decline in the incidence of invasive Hib infections. But, the invasive diseases are still reported, particularly nontypeable H. influenzae (noncapsulated) remain as an important pathogen. However, there is no evidence that nontypeable H. influenzae infections have increased in frequency. Nontypeable H. İnfluenzae serotype is encountered as a cause of acute bacterial meningitis among all ages. In this paper, we present to draw attention to the causative bacterium, in a case of bacterial meningitis caused by nontypeable H. influenzae infection in a child immunized with Hib vaccine.

Nontypeable Haemophilus Influenzae; Meningitis; Child

Corresponding Author: Deniz Aygün, Çocuk Sağlığı ve Hastalıkları AD, Enfeksiyon Hastalıkları, Klinik İmmünoloji ve Allerji BD, İstanbul Üniversitesi, Cerrahpaşa Tıp Fakültesi, 34098, İstanbul, Türkiye. GSM: +905327868682 F.: +90 2126328633 E-Mail: fdenizaygun@gmail.com

#### Giris

Haemophilus influenzae, sadece insanda hastalık yapan, küçük, pleomorfik, gram negatif, fakültatif, anaerop bir kokobasildir. Dış yüzeyinde bulunan polisakkarit yapıdaki kapsülü mikroorganizmayı fagositozdan korur. Polisakkarit kapsül varlığı H. influenzae'nın tür serotip sınıflamasında önemlidir. Kapsüllü olanlar serolojik olarak altı serotipe [a,b,c,d,e,f] ayrılır. Kapsül içermeyen türler ise tiplendirilemeyen olarak adlandırılır. Tip B kapsül ribosil ve ribosil fosfat polimerinden oluşur ve polisakkarit asının en önemli antijenik bileşenidir [1]. Solunum vollarında kolonize olan H. influenzae'nın tiplendirilemeyen [kapsülsüz] suşları sıklıkla bronşit, otitis media, sinüzit, konjunktivit ve idrar yolu enfeksiyonu gibi mukozal hastalıklara neden olur. Menenjit, pnömoni, epiglottit, osteomyelit, septik artrit, sepsis gibi invaziv enfeksiyonlardan ise çoğunlukla [%95] tip b kapsüle sahip olan H. influenzae sorumludur [2]. Olguların %85'i beş yaş altı çocuklardır. Hib konjuge aşısının yapılmasıyla invaziv Hib hastalığı geçirme sıklığı anlamlı olarak azalmıştır. Aşılamaya karşın halen invaziv hastalık bildirimleri olmaktadır ve bu olgular arasında kapsülsüz [tiplendirilemeyen] serotiplere bağlı enfeksiyonlar göreceli olarak daha sık görülmektedir. Ancak tiplendirilemeyen serotiplerin neden olduğu invaziv hastalık sıklığında artış ile ilgili henüz kesin bir veri yoktur. Bu yazıda tiplendirilemeyen H. influenzae'nın Hib aşısı yapılmış, sağlıklı çocuklarda da menenjite neden olabilen potansiyel bir patojen olarak akılda tutulması gerektiği vurgulamaktadır.

#### Olgu Sunumu

Aşıları Sağlık Bakanlığı Aşı Takvimine göre düzenli olarak yapılmış altı yaşında kız hasta ateş, kusma ve şiddetli baş ağrısı nedeniyle başvurdu. Fizik muayenesinde boy: 115 cm [50.p], kilo: 23 kg [50.p], kan basıncı 100/60 mmHg, vücut sıcaklığı: 39°C, bilinci açık, meningeal irritasyon bulguları pozitif olarak saptandı. Diğer sistem muayeneleri doğaldı. Laboratuvar incelemelerinde lökositoz, nötrofili, lenfopeni [19000/mm3, nötrofil: 17600, lenfosit: 1400], anemi [hemoglobin: 10.9 g/L] vardı. C-reaktif protein: 5.6 mg/dl, eritrosit sedimantasyon hızı 22 mm/saat olarak saptandı, diğer serum biyokimya ve elektrolit düzeyleri normaldi. Hastanın immunglobulin G, A ve M düzeyleri yaşına uygundu. Beyin omurilik sıvısının [BOS] rengi bulanık, BOS biyokimyasında glukoz düşük [14 mg/dl, eş zamanlı kan şekeri: 121 mg/dl]), protein yüksek [275 mg/dl] saptandı, 4000 nötrofil /mm3 görüldü. Hastanın kan kültürü ve BOS kültürü alınarak vankomisin ve seftriakson tedavileri başlandı. Tedavinin 3. gününde ateş yakınması ve meningeal iritasyon bulguları geriledi, kontrol lomber ponksiyonunda BOS berraktı, biyokimyası normaldi ve hücre saptanmadı. BOS kültüründe tiplendirilemeyen Haemophilus influenzae üremesi oldu. Hastanın tedavisi 14 güne tamamlandı, komplikasyon gelişmedi, hasta șifa ile taburcu edildi.

#### Tartışma

Çocukların üçte biri 18 ayına gelmeden H. influenza'nın hem kapsüllü hem de kapsülsüz suşları ile kolonize olmaktadır. İndeks vakanın olduğu durumlarda taşıyıcılık oranı kreş ve gündüz bakım evlerinde %60'a kadar çıkmaktadır. Solunum yollarında kolonize olan H. influenzae transmural göç veya bağımsız hücreler arası mekanizma ile subepitelyal boşluğa ulaştıktan sonra

kan dolaşımına katılarak invaziv hastalığa neden olmaktadır. İnvaziv hastalık özellikle kapsüllü H. influenzae tip b ile oluşmaktadır. Olgular büyük çoğunlukla beş yaşın altında özellikle de 6-12 avlık cocuklardır.

Tiplendirilemeyen H. influenzae serotipleri kapsülü olmasa da HMW1 ve HMW2 gibi yapışma faktörleri, IgA 1'i parçalayan proteaz salınımı, peptidoglikan ve lipopolisakkaritlerin silyalara inhibitör etkisi sayesinde sağlıklı çocuklarda da invaziv hastalığa neden olmaktadır.

Arkansas'ta yapılan bir çalışmada, tiplendirilemeyen H. İnfluenzae'nın invaziv hastalıklara neden olduğu çocukların % 86'sı 4 yaş altında iken, % 68'ininde ise alta yatan hastalığı saptanmıştır [4]. Olguların 4 yaş altında daha sık olması yüksek nazofarengeal kolonizasyon ile açıklanmaktadır. Ayrıca bu yaş grubunda viral solunum yolu enfeksiyonlarının fazla olması mukozal inflamasyonu ve bakteri invazyonunu kolaylaştırmaktadır. Tiplendirilemeyen H. influenzae'nın neden olduğu invaziv hastalıklarda travma, immun yetersizlik veya beyin omurilik sıvısı kaçağı gibi allta yatan hastalıkların araştırılması gerekmektedir. Bizim olgumuz daha önce sağlıklı olan aşıları tam olarak yapılmış 6 yaşında kız çocuğuydu. Olgumuzun kantitatif ımmunglobulin düzeyleri normal bulundu.

1985 yılında konjuge Hib aşısının ABD'de kullanıma girmesinden sonra invaziv hastalık insidansı belirgin olarak azalmıştır ve beş yaş altında 1/100 000'in altına inmiştir [2]. Avrupa'da ise son on yıl içinde aşı sonrası Hib menenjiti görülme sıklığının %90 azaldığı bildirilmiştir [3]. Endonezya'da yapılan bir çalışmada ise Hib aşısı ile her 179 çocuktan birinde menenjit, her 18 çocuktan birinde ise pnömoni gelişimi engellenmiştir [5].

Aşılama sonrası invaziv Hib enfeksiyonlarının azaldığı kanıtlanmıştır ancak az da olsa aşı suşundan farklı tiplerdeki suşlar veya tiplendirilemeyen H. İnfluenzae suşları ile invaziv enfeksiyonlar bildirilmektedir [6]. Health ve arkadaşları aşılama sonrası tiplendirilemeyen serotiplerde artışa dikkat çekmişlerdir [7]. Adderson ve arkadaşları ise H. influenzae tip a'nın neden olduğu dört menenjit vakası bildirmişlerdir [8]. Aşılamaya rağmen tiplendirilemeyen Hib enfeksiyonlarının görülmesinin nedeni olarak tek bir kapsül serotipine karşı aşılama, kapsül yapısındaki değişiklikler, genotip farklılık gibi fikirler öne sürülse de ispatlanmış bir kanıt bulunamamıştır [6]. Ancak tiplendirilemeyen serotiplerin neden olduğu invaziv hastalık sıklığında artış ile ilgili henüz kesin bir veri yoktur.

Bu yazı tiplendirilemeyen Haemophilus influenzae'nın altta yatan bir hastalığı olsun olmasın her yaş grubunda akut bakteriyel menenjite yol açan etkenler arasında hatırlanması gerektiğini vurgulamak amacıyla yazılmıştır.

## Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı beyan edilmemiştir.

#### Kaynaklar

- 1. St Geme JW 3rd, Takala A, Esko E, Falkow S. Evidence for capsule gene sequences among pharyngeal isolates of nontypeable Haemophilus influenzae. J Infect Dis 1994;169(2):337-42.
- 2. American Academy of Pediatrics. Haemophilus influenzae Infections. In: Pickering LK, Baker CJ, Long SS, McMillan JA (eds). Red Book: 2006 Report of the Committee on Infectious Diseases. 27th edition. Elk Grove Village, IL: American Academy of Pediatrics, 2006.p.310-8.
- 3. Bloom DE, Canning D, Weston M. The value of vaccination. World Economics 2005;6(3):15-39.

- 4. O'Neill JM, St Geme III JW, Cutter D, Adderson EE, Anyanwu J, JacobsRF, et al. Invasive disease due to nontypeable Haemophilus influenzae among children in Arkansas. J Clin Microbiol 2003;41(7):3064-9.
- 5. Gessner BD, Sedyaningsih ER, Griffiths UK, Sutanto A, Linehan M, Mercer D, et al. Vaccine-preventable haemophilus influenza type B disease burden and cost-effectiveness of infant vaccination in Indonesia. Pediatr Infect Dis J 2008;27(5):438-43.
- 6. Ulukol B. Invasive haemophilus influenzae type b infections and haemophilus ınfluenzae type b vaccıns. Türkiye Klinikleri J Pediatr Sci 2007;3(11):55-64.
- 7. Heath PT, Booy R, Azzopardi HJ, Slack MP, Fogarty, et al. Non-type b Haemophilus influenzae disease: clinical and epidemiologic characteristics in the Haemophilus influenzae type b vaccine era. Pediatr Infect Dis J 2001(20):300-5.
- 8. Adderson EE, Byington CL, Spencer L, Kimball A, Hindiyeh M, Carroll K, et al. Invasive serotype a Haemophilus influenzae infections with a virulence genotype resembling Haemophilus influenzae type b: emerging pathogen in the vaccine era? Pediatrics 2001;108(1):1-6.

#### How to cite this article:

Aygün D, Sinoplu AB, Çakırlar FK, Çokuğraş F, Camcıoğlu Y. Non-Type B Haemophilus Influenzae Meningitis: A Case Report. J Clin Anal Med 2015;6(suppl 5): 715-7.

## **Dressler Syndrome**



#### Dressler Sendromu

Dressler / Dressler

Erkan Ceylan

Göğüs Hastalıkları A.D. Medeniyet Üniversitesi Tıp Fakültesi, İstanbul, Türkiye

#### Özet

Dressler Sendromu(DS), plevra ve perikardın inflamatuvar reaksiyonuna sekonder gelişen ateşli bir hastalıktır. Sıklıkla perikard operasyonu yapılan hastalarda görülür. Ancak, DS miyokard infarktüsünü takiben, perkütan işlemlerden sonra sıra dışı bir komplikasyon olarak gözlenebilir. Koroner stent implantasyonu sonrası, epikardiyal pacemaker sonrası, transvenöz pacemaker sonrası, künt travma sonrası, bıçak yaraları ve kalp ponksiyonları sonrası gözlenmiştir. Perikardiyal efüzyon sıklıkla sendroma eşlik eder ve postoperatif erken veya geç dönemde kardiyak tamponad gelişebilir ve hatta tekrarlayan kardiyak tamponadlarda gözlenebilir. Sendrom perikardiyal ve/veya plöretik ağrı, plevral efüzyon, pnömoni, anormal EKG ve radyografi bulguları ile karakterizedir

#### Anahtar Kelimeler

Dressler Sendromu; Post Kardiyak İnjuri Sendromu; Plevral Efüzyon; Perikardiyal Efüzyon

#### Abstract

Dressler Syndrome (DS) is a febrile illness secondary to an inflammatory reaction involving the pleura and pericardium. It is more common in patients who have undergone surgery that involves opening the pericardium. However, DS has also been described following myocardial infarction and as an unusual complication after percutaneous procedures such as coronary stent implantation, after implantation of epicardial pacemaker leads and transvenous pacemaker leads, and following blunt trauma, stab wounds, and heart puncture. Pericardial effusions often accompany the syndrome and may develop into early or late postoperative cardiac tamponade and even recurrent cardiac tamponade. The syndrome is also characterized by pericardial or pleuritic pain, pleural effusions, pneumonitis, and abnormal ECG and radiography findings.

#### Keywords

Dressler Syndrome; Postcardiac Injury Syndrome; Pleural Effusion; Pericardial Effusion

DOI: 10.4328/JCAM.3939 Received: 04.10.2.105 Accepted: 30.10.2015 Printed: 01.10.2015 J Clin Anal Med 2015;6(suppl 5): 718-23 Corresponding Author: Erkan Ceylan, Zümrütevler Mah. Pirsultanabdal Cad. Nishadalar Sitesi 56/7 Maltepe, İstanbul, Türkiye. E-Mail: drerkanceylan@yahoo.com

#### Giris

Dressler sendromu (DS); Postakut myokardiyal sendrom, post kardiyak injuri sendromu, postperikardiyotomi sendromu olarak ta tanımlanmaktadır. DS perikard ve/veya miyokard hasarlanmasını takip eden, haftalar içinde gelişen ateş, plöröperikardit ve parankim infiltrasyonu ile karakterize bir klinik tablodur [1-3]. Bu sendrom literatürde miyokard enfarktüsü, kardiyak cerrahi, künt göğüs travması, perkütan sol ventrikül biyopsisi, pacemaker yerleştirilmesi, koroner stent implantasyonu, kalp ponksivonları ve anjiyoplasti sonrası tanımlanmıştır [1,4].

#### **Tarihce**

DS ilk olarak 1953 yılında mitral kapak kommissurotomi operasyonu geçiren hastalarda Soloff tarafından gözlenmiş ve postkommissurotomi sendromu olarak tanımlamıştır [5]. Soloff postkommissurotomi sendromunu etyolojik nedeninin romatizmal ateş reaktivasyonu olduğun savunmuştur. Itoh 1958'de bu klinik tabloyu postperikardiyotomi sendromu olarak tanımlarken, sendromun sıklığını da %10-50 arasında olduğunu gözlemlemiştir [6-9]. Günümüz bilgileri ile uygun tanımlama 1956'da Dressler tarafından yapılmış ve DS olarak adlandırmıştır [1].

#### İnsidans:

Dressler, myokart infarktüsü (ME) sonrası DS insidansını %3-4 olarak bildirirken, Light, ME ve perikarditli olgularda %15 olarak bildirilmiştir [10,11]. Engle ve arkadaşları 257 çocuk olguda, kardiyak cerrahi sonrası DS insidansını %30, Miller ve arkadaşları ise 1 yıllık periyotta kardiyak cerrahi geçiren 944 olguda %17.8 olarak belirtmişlerdir [9,12].Birçok araştırmacı ise DS insidansını %5'den az gözlemişlerdir [13-17]. Miller ve arkadaşları kardiyak cerrahi geçiren olgulardaki DS insidansını diğer tüm cerrahi geçirenlerle yaklaşık olarak eşit oranda ortaya çıktığını belirtmiştir [12].

## Predispozan Faktörler ve Patogenez:

Sendromun kesin etyolojisi bilinmemekle beraber perikardın hasar görmesi sonrası açığa çıkan antijenlere karşı oluşan otoantikorların oluşturduğu otoimmün bir olay olduğu kabul edilmektedir. Transplantasyon sonrası postperikardiyotomi sendromu gelişen hastalarda yapılan bir çalışmada aktive T helper (CD4+/25+) ve sitotoksik T-hücrelerinin(Leu-7+/CD8+) arttığı gözlenmiştir [18,19].

Dressler, sendromun hipersensitivite reaksiyonuna bağlı olabileceğini bildirirken[1], Janton; romatizmal ateş reaktivasyonuna [20], Ito; travmatik perikardite [6], Burch ve Engle Coxsackie B gibi bir latent virüs enfeksiyonuna bağlı olabileceğini bildirmiştir [21,22]. Farklı epidemiyolojik çalışmalarda toplumda enfeksiyon prevalansının yüksek olduğu dönemlerde DS'nin yüksek insidansa sahip olduğu gösterilmiştir [23-25]. Bu çalışmalarda viral enfeksiyonların immün cevabı tetiklediği düşünülmüştür. Engle ve arkadaşları antikalp antikoru mevcut olan DS sendromlu olgularda yüksek viral antikor titreleri saptamış ve virüs enfeksiyonlarının DS etyolojisinde önemli rolü olduğunu belirtmişlerdir [26]. Üst solunum yollarında viral enfeksiyonu olan 150 hastada yapılan prospektif bir çalışmada sporadik olarak gelişen myoperikardit ve Q dalga infarktı gösterilmiştir. Ancak, perikardiyal veya plevral sıvılarda virüs izole edilmemiştir [27].

Günümüzde sendromun nedeni tam olarak bilinmemekle beraber immünolojik bir temele dayandığı bilimsel olarak kabul edilmektedir. Perikard hasarı, yatkınlık gösteren bireylerde immünolojik olayları başlatabilmektedir. Perikarda cerrahi işlem uygulanan olgularda, sendromun gelişimi ile antikalp antikorları

arasında yakın ilişki gösterilmiştir [9-11].Benzer şekilde koroner arter bypass cerrahisi sonrası DS gelişen olgularda aktin ve miyozine karşı anlamlı antikor seviyeleri saptanmıştır [24]. DS'de miyokard hasarı veya ME sonrası salınan miyokardiyal antijenlere karşı gelişen immün aktivasyon lokal inflamatuvar reaksiyonu tetikleyerek plevra, sinovya gibi uzak organları etkilemektedir [28,29]. Miyokard hasarından DS ortaya çıkana kadar 2-3 haftalık latent dönemde immün reaksiyon gelişmektedir [30]. Kortikosteroid veya nonsteroid antiinflamatuar ilaçlara(NSAİİ) iyi yanıt vermesi ve relapslara gözlenmesi DS patogenezinde immünolojik mekanizmaların önemli bir kanıtıdır. Bununla birlikte miyokard hasar derecesinin DS ile direkt ilişkili olmaması da sendromun immünolojik özelliğini desteklemektedir [24,31].

#### Risk Faktörleri:

Günümüzde çok çeşitli risk faktörleri tanımlanmıştır. Bunlar; genç yaş, perikardit, aort kapak replasman öyküsü, kortikosteroid kullanımı, BRh(-) kan grubu olma ve daha önce halotan, enfloran anestezisi alma bulunmaktadır. Bununla beraber Mayıs, Haziran, Temmuz aylarında sendrom sıklığının arttığı tespit edilmiştir [12, 29]. Bu risk faktörlerinin bilinmesi DS'nin erken tanısı için çok değerli bilgiler verebilir.

#### Klinik Özellikler:

DS kardiyak travma veya bir ME sonrası ortaya çıkan ateş, göğüs ağrısı, perikardit, plörit ve pnömoni ile karakterizedir. Semptomlar ME'yi takiben ilk haftada çıkabilmekle beraber sıklıkla 2. veya 3. haftada gözlenir. Kardiyak operasyonlar sonrası ise semptomlar takip eden ilk 3 haftada gözlenir; fakat 3 gün ve 1 yıl arasında herhangi bir zamanda da ortaya çıkabilir [11,32,33,34]. Kardiyak cerrahiyi takiben olguların %10-%50'sinde ortaya çıkabileceği ve sendromun başlangıcının pacemaker implantasyonunu takiben diğer durumlardan daha hızlı olduğu bildirilmektedir [12]. Bajaj ve arkadaşları 68 yaşında bir erkek hastada pacemaker takılmasını takiben bir iki gün içerisinde DS geliştiğini belirtmişlerdir[35].

Sendromun iki kardinal semptomu ateş ve göğüs ağrısıdır. Göğüs ağrısı sıklıkla ateş başlamadan önce vardır. Ağrı; genellikle baskı tarzında, künt-yanıcı, miyokardiyal iskemiyi taklit edici özelliklerdedir. Plöretik nitelikte olabilirken göğüs ağrısı bıçak saplanır tarzda omuz ve skapulalar arası bölgeye yayılabilir. Derin inspirasyonla veya gövdenin döndürülmesiyle artar, oturur pozisyonda veya öne eğilirken azalır. Ağrı başlangıcından sonra saatler veya günler içinde plevral sürtünme sesi duyulabilir. Plevral sürtünme sesi pozisyonla veya solunumla değişebilir, geçici olduğu için seri oskültasyonlar yapılmalıdır [36]. Hemen hemen tüm olgularda perikardiyal frotman vardır ve çoğunda da perikardiyal efüzyon mevcuttur. Olguların yaklaşık %75'inde lineer ya da yama tarzında ve genellikle akciğer bazalinde lokalize pulmoner infiltratlar gözlenir [24]. Laboratuvar incelemesinde genellikle 10-20 bin arasında lökositoz ve orta derecede artmış sedimentasyon hızı(ESR) saptanır [11,33,34]. DS'de plevral tutulum sık gözlenir. Çalışmalarda %68-%83 oranında plevral efüzyon saptanmıştır [10,37]. Genel olarak plevral efüzyon az miktarda olup, baskın klinik bulgu perikardite bağlıdır. Perikardiyal efüzyon miktarı genellikle azdır ve tamponad nadirdir. Plevral efüzyonlar unilateral yada bilateral görülebilmektedir [37]. Stelzner ve arkadaşlarının 35 DS'li olgunun klinik ve laboratuvar bulgularını değerlendirdikleri çalışmalarında vakaların %96'sında ESR'de yükselme, %91'inde plevral efüzyon, %66'sında ateş, %63'ünde perikardiyal frotman, %57'sinde dispne, %51'inde raller, %49'unda lökositoz ve %46'sında plevral frotman tespit

etmişlerdir. Plevral efüzyon saptanan 16 olgunun 12'sinde efüzyonun eksuda vasfında olduğu gösterilmiştir [37]. Hastaların kliniklerinde öksürük, ateş gibi pnömoni bulguları, fizik muayenelerinde de pnömoni, plevral sıvı ve perikardiyal sıvıya sekonder muayene bulguları gözlenir. Sistemik sıvı retansiyonu ve hepatomegali ve hipoksemi gözlenebilir.

## Komplikasyonlar:

Kardiyak Tamponat: DS'ye bağlı kardiyak tamponat yaşamı tehdit eden önemli bir komplikasyon olup literatürde %0.1-6 oranlarında bildirilmiştir [38,39,33]. Perikardiyal efüzyon acil perikardiyosentez ve drenaj gerektirir. Açık kalp ameliyatı olan 1290 hastalık prospektif bir çalışmada da, DS tanısı alan 10(%0.8) hastada tamponat gelişmiştir [38]. Bu hastalardan biri sodyum warfarin ve ikisi ise aspirin alıyordu. Bu hastalardan 9'una perikardiyosentez birine ise tekrarlayan tamponat nedeniyle perikardiyal stripping uygulandı. Olgularda ölüm gözlenmedi. Birçok çalışmada gösterilmiştir ki DS'de tamponat nadirdir ve eşzamanlı antikoagülan tedavi olmadan da ortaya çıkabilir. Tek antikoagülan kullanımı çoğu durumda güvenli olmakla beraber bu hastalar yakından izlenmelidir. Diğer taraftan antikoagülan tedavi ile az dahi olsa tehlikeli tamponad gelişebilir. Bu nedenle antikoagülan tedavi prostetik kapak, geniş miyokard infarktüsü(özellikle anterior), atriyal fibrilasyon gibi yüksek riskli hastalarla sınırlı olmalıdır [10,40-42,43].

Konstrüktif Perikardit: Postoperatif dönemde olguların %5'inden azında gözlenir. Konstrüktif perikardite perikard kalınlaşır ve kalbe yapışır. Bu halde ventriküllerin dolması kısıtlanır. Bu olgularda perikardiyotomi gerekli olabilir. Bu olgularda inflamatuvar yanıta bağlı gelişen göğüs ağrıları solunumsal sıkıntılara ve hipoksemi gelişimine neden olabilir. Bu nedenle pulse oksimetre ile sürekli oksijen satürasyonu takibi yapılmalıdır [44].

Koroner Arter ve Koroner Arter Bypass oklüzyonu: Urschel ve arkadaşları kalp cerrahisi sonrasında perikardiyal efüzyon saptanan 45olgudan 10'unda greft oklüzyonu saptamışlar ve bunlardan 4'ü yaşamını yitirmiştir [45]. Başka bir çalışmada kalp ameliyatları sonrasında perikardiyal efüzyonlu hastalarda, greft oklüzyonu ve perikardiyal efüzyonun ağırlığı arasında herhangi bir korelasyon saptanmamıştır [39].

Pulmoner Tutulum: En yaygın tutulum şekli plevral efüzyondur(%62). Bununla beraber araştırmaların birçoğu akciğer infiltratları üzerine odaklanmıştır. Parankim infiltrasyonu ise %50 olguda gösterilmiştir. Radyolojik bulgular değişik şekilde yorumlanmakla beraber sıklıkla konjestif kalp yetmezliği ve akciğer enfeksiyonları ile karışabilmektedir [9,46].

## Tanı:

DS'nin tanısal spesifik bir bulgusu olmadığı gibi kesin bir tanı yöntemide bulunmamaktadır. ME veya kardiyak işlem/operasyonu takiben oluşmuş bir plevral efüzyon, özellikle perikardit bulguları ile birlikte gelişen bir klinik varlığında DS düşünülmelidir. Serum ve plevral sıvı laboratuvar değerleri ayırıcı tanıda değerli olmakla birlikte uygun klinik ve laboratuvar sonuçları sonrasında pnömoni, kalp yetmezliği, pulmoner emboli tanılarının ekarte edilmesi ile kesin tanı konulabilir. Literatürde DS gelişimi için bildirilen Mayıs, Haziran, Temmuz aylarında bulunma, 54 yaşın altında olma, daha önce perikardit geçirilmesi veya aort kapak replasmanı uygulanması, enfloran veya halotan anestezisi verilmesi ve prednizon kullanılması gibi durumlarda DS gelişme olasılığı daha yüksektir [12, 29, 47].

DS tanısında bazı kriterler kullanılmaktadır [36]. Bunlar major ve minör kriterler olarak sınıflandırılmıştır. Major kriterler; perikardiyal veya plevral frotman, göğüs ağrısı ve 380'nin üzerinde ateş olmasıdır. Minör kriterler ise; ESR, C reaktif protein (CRP) seviyesinde ve lökosit sayısında artış olmasıdır. Pnömoni, kalp yetmezliği, pulmoner emboli vb. durumlar ekarte edildikten sonra iki major ve bir minör kriter varlığının DS tanısı için yeterli olduğu ifade edilmektedir [32].

DS'de en sık görülen semptomlar plöretik göğüs ağrısı (%91)ve nefes darlığıdır (%57). En sık bulgular ateş (%66), plevral frotman (%46), akciğer muayenesinde raller (%51) ve perikardiyal frotmandır (%63). En sık gözlenen laboratuvar bulguları ESR'de artış (%96) ve lökositozdur (%49). Hastaların akciğer grafisinde en sık plevral efüzyon saptanmaktadır (%83) [37].

Olguların üçte ikisinde plevral sıvı gözlenmekte olup, sıvı sıklıkla bilateraldir ve çoğu vakada az miktardadır. Plevral sıvı eksuda niteliğinde olup, glikoz ve pH değerleri normaldir. Bazı olgularda pH 7,4 den büyük olabilir. Plevral sıvı, olguların %30'unda hemorajik görünümdedir. Plevral sıvıda hücresel dağılım değişkendir, polimorfonükleer lökositler veya mononükleer hücreler akut dönemde yüksek değerlerde saptanırken, kronik vakalarda lenfosit hâkimiyeti gözlenebilir [11,37,48]. DS'de gelişen perikardiyal efüzyon ise genelde az miktarda olup, plevral sıvı içeriğine benzer özellik taşır. Perikard tamponadına neden olması son derece nadirdir [49].

Kan sayımında lökositoz ve sola doğru bir kayma gözlenebilir. Kan kültürleri inflamatuvar hastalıklarla enfeksiyon hastalıklarının ayırımı, erken tanı ve ayırıcı tanıda önemlidir. Bu nedenle kan kültürlerinin negatif olduklarının gösterilmesi önemlidir. ESR ve CRP gibi akut faz reaktanları yüksek bulunmaktadır. Antikalp ve antikardiyolipin antikor seviyelerinin yüksekliği tanıda kullanılabilmekle beraber, sensitivitesinin düşük, spesifitesi ılımlı yüksektir [50]. Klinik çalışmalarda antikalp antikorları ile DS gelişimi arasında doğru ilişki olduğu gösterilmiştir [51,52]. DS'li hastaların serumlarında antimiyokardiyal antikor titrelerinin yüksek olduğu gösterilmiştir [53]. Benzer şekilde plevral sıvıda antimiyokardiyal antikor seviyelerinin yüksekliği gösterilmiş ve buna bağlı olarak plevral sıvıda antimiyokardiyal antikor titresinin ölçümünün tanısal değeri olabileceği, ayrıca plevral sıvıda kompleman seviyesinin düşük olmasının da tanıda önemli olabileceği bildirilmiştir [54,55]. Kalp enzimleri ölçümü, sonuçlarının çok farklı değerlerde gelmesi nedeniyle genellikle yararlı değildir.

Ayrıca DS'li olgularda hemoptizi, artrit, artralji ve nedeni açıklanamayan anemi gibi bulgular da görülebilmektedir [32]. Ancak bu klinik ve laboratuvar bulguların hiçbiri DS için spesifik değildir. Hemorajik perikardiyal veya plevral eksudatif efüzyon varlığı DS'yi düşündürse de patognomik değildir. Bu nedenle DS tanısı tipik klinik bulgulara ek olarak pulmoner emboli, konjestif kalp yetmezliği, atelektazi ve pnömoni tanılarının ekarte edilmesiyle konur [56,57]. Pulmoner embolinin kesin ekartasyonu önemlidir, çünkü antikoagülan tedavi yaklaşımı postkardiyak injuri sendromunda kontrendikedir ve bu olgular hemoperikardiyum gelişimi açısından risk altındadır [1,3,58]. Kalp yetmezliği anamnezinin olmaması, normal ekokardiyografik inceleme, eksudatif plevral sıvı tespiti ile kalp yetmezliği ekarte edilir. Pürülan balgam olmaması, akciğer fizik muayene bulgularının azlığı ve pnömonik konsolidasyonun küçüklüğü, yada olmaması, pnömoni ile orantısız ağır klinik tablo varlığı DS lehine yorumlanır. Kan ve plevral sıvı kültürlerinde üreme olmaması ile parapnömonik efüzyon dışı tanılar klinik olarak önem kazanır.

Göğüs radyografisi: Pulmoner infiltraların eşlik ettiği plevral efüzyon varlığında akciğer grafisi tanıda yararlıdır. Kalp silueti perikardiyal kese içinde bulunan sıvı miktarı ile orantılı olarak büyüktür.

Elektrokardiyografi: Elektrokardiyografik bulgular sıklıkla anormaldir. Başlangıç bulgular, global ST segment elevasyonu ve T dalga inversiyonu ile perikarditi taklit edebilir. Miyokard inflamasyonundan kaynaklanan subepikardiyal yaralanma, ST segment elevasyonuna neden olur. Büyük perikardiyal efüzyonlar, düşük QRS amplitüdüne neden olabilirler.

Ekokardiyagrafi: Ekokardiyografi standart tanısal bir testtir. Radyografiye göre çok daha hassas bir görüntüleme yöntemidir. DS'nin erken evrelerinde, küçük miktarlardaki sıvı sistolde sol ventrikülün posterior kısmında tespit edilebilir. Artmış sıvı miktarlarını ise ekokardiyografi ile saptama çok daha kolaydır. Konjestif kalp yetmezliğini DS'den ayırt etmede ekokardiyografi yardımcıdır. Kardiyak out put her iki durumda da azaltılır. Büyük efüzyonlu DS'de bir veya birden fazla kalp boşlukları perikard sıvısı ile sıkıştırılmış olabilir. Ekokardiyografi ventriküler kontraktilite değerlendirilmesinde özellikle yararlıdır.

Manyetik Rezonans Görüntüleme: Kardiyak manyetik rezonans görüntüleme kalp dinamikleri ve perikard anormallikleri değerlendirmek için daha sık kullanılmaya başlanmıştır. Bu görüntüleme yöntemi transtorasik ekokardiyografi ile kolayca görüntülenemeyen loküle sıvılar ve posterior perikard sıvısı koleksiyonlarını belirlemede daha yararlıdır.

#### Ayırıcı Tanı

- Pulmoner Emboli
- Pnömoni
- · Şilöz perikardiyal efüzyon
- Şilöz plevral efüzyon
- · Konstrüktif Perikardit
- · Konjestif Kalp Yetmezliği
- · Bakteriyel Endokardit
- · İnfektif Perikardit
- · Nonviral Myokardit
- · Viral Myokardit

#### Tedavi:

Özellikle perikardit bulguları ile birlikte gelişen bir klinik tablo varlığında hemodinamik takip, pulmoner anjiyografi, ventilasyon sintigrafisi, kültür sonuçları ile (balgam, plevra sıvısı, perikard sıvısı, kan) diğer tanıların kesin ekartasyonu sonrası tedavi başlanır. Tarihsel süreçte Dressler, etyolojide immün reaksiyonları sorumlu tutarak kortikosteroid tedavisinin başarılı olacağını, antibiyotik tedavisi başarılı olmayacağını belirtmiştir [1]. Weiser ve arkadaşları ise pulmoner infiltratları kalp yetmezliğine bağlamış ve agresif kalp yetmezliği tedavisi önermiştir [59]. DS süphesi olan hastalar hemodinamik olarak stabilse genellikle tetkik ve tedavileri ayaktan devam edebilir. Semptom ve bulguları kardiyak tamponadı düşündüren olgular, genel durumu ve hemodinamisi bozuk ciddi olgular hastaneye yatırılarak tetkik ve tedavi edilmelidirler. Bu olgularda perikardiyal efüzyon drenajı ve klinik belirtilerin iyileşmesi sonrası ayaktan tedavi edilebilirler.

DS'nin tedavisinde hastaların çoğu NSAİİ ve/veya kortikosteroidlere iyi yanıt verir. Genellikle aspirin, indometasin, ibuprofen gibi NSAİİ sıvı azalmasını takiben 4-6 hafta süreyle verilirler. NSAİİ yeterli ve güvenli dozlarda hastaya verilmelidir. Antiplatelet etkininin ön planda olması gereken olgularda aspirin daha öncelikli düşünülen tedavi olmalıdır. NSAİİ'le genelde iyi sonuçlar alınırken bazı hastalarda kortikosteroidler gerekebilir [60]. Kortikostroidler yaygın yan etkileri nedeniyle daha kısıtlı kullanılmaktadır. NSAİİ intoleransı olan, kliniği şiddetli olan, tedaviye

rağmen relaps gösteren yada bu ilaçlarla yanıt alınamayan olgularda kortikosteroidler önerilir. Kortikosteroidler orta-yüksek dozlarda kullanılabilirler. Genellikle 60mg/gün başlayıp haftada 5mg'lık doz azaltma şeklinde 1-4 hafta süreyle kullanılırlar. Kortikosteroidler, klinik belirtilerde hızlı düzelme ve antikalp antikorlarda hızlı düşme sağlar [24,61].

DS'li hastalarda NSAİİ'lerin karşılaştırıldığı çift-kör plasebo kontrollü randomize bir çalışmada 10 gün süre ile indometazin (25mg 4x1) veya ibuprofen (600mg 4x1) uygulanmış ve indometazin verilen olguların %90,7'sinde, ibuprofen verilen olguların ise %87,5'inde semptomların kaybolduğunu belirtilmiştir. Her iki grupta ilaç yan etkileri, hastanede kalış süreleri ve iskemi insidansının benzer olduğu gözlenmiştir [62].

Koroner arter bypass cerrahisi geçiren olgularda DS tanısını net olarak koymak çok önemlidir. Çünkü perikardit, graft oklüzyonuna neden olabilmektedir. Urschel ve arkadaşları koroner arter bypass operasyonundan sonra sendrom gelişen ve semptomatik olarak tedavi gören 14 olgunun 12'sinde (%86) graft oklüzyonu ortaya çıktığını, prednizolon ile tedavi edilen (30mg/gün 1 hafta ve azaltılarak 5 haftada kesilen dozda) ve ilave olarak 600 mg/ gün aspirin verilen 31 olgunun sadece 5'inde (%16) graft oklüzyonu geliştiğini belirtmişlerdir [45].

Tekrarlayan perikardiyal efüzyonda puşe yüksek doz intravenöz immünglobulin ve haftalık düşük doz metotreksat kullanımının başarılı sonuçları tanımlanmıştır [63,64]. Rekürrens durumunda tedavide Kolşisin kullanımı tavsiye edilmektedir [66].

Perikardiyosentez: DS'nin neden olduğu tamponad hayatı tehdit eden bir durumdur. Postperikardiyotomi sendromunda görülen inflamatuvar değişiklikler perikardta lokal plevral sıvı koleksiyonları ve perikardiyal yapışıklıklara neden olabilir. Solunum sıkıntısına neden olabilen geniş plevral efüzyonlu olgularda terapötik torasentez yapılmalıdır. Benzer şekilde yaşamı tehdit eden perikardiyal efüzyonların neden olduğu tamponat durumunda perikardiyosentez uygulanmalıdır [57].

Perikardiyosentezde standart subksifoid yaklaşım önerilir. Ekokardiyografi eşliğinde lokalize tampon bölgesi gözlemlenir. Genişletilmiş kateter drenajı ile ekokardiyografi eşliğinde perikardiyosentez perikardiyal efüzyonlu hastalarda primer yöntemdir. Tüp drenajı genellikle antiinflamatuar tedavi başlatıldıktan itibaren 24-48 saat boyunca takılı bırakılır.

Diyet ve aktivite: Hastalarda genellikle iştah azalmıştır. Ancak, spesifik diyet kısıtlamaları genellikle gerekli değildir. Yorucu, efor gerektiren faaliyetlerden kaçınmalıdırlar. Tedavide sadece yatak istirahati hafif vakalarda yeterli olabilir. Ateş düşüp, akciğer grafisi ve EKG bulguları düzelene kadar sıkı yatak istirahati uygulanmalıdır.

Cerrahi Tedavi: Cerrahi olarak perikardiyal pencere açılması persistan semptomları olan veya medikal tedaviye rağmen relaps gözlenen hastalarda gerekli olabilir. Bu açık bir torakotomi ile veya VATS (video yardımlı torakoskopik cerrahi) ile yapılmaktadır [63, 64]. Perkütan balon perikardiyotomide bu hastalar için bir başka alternatiftir. Bu işlem floroskopi altında balon kateter kullanarak kateterizasyon laboratuvarında oluşturulduğu için daha az invazif bir işlemdir [57, 66].

#### **Prognoz**

DS vakalarının çoğu birkaç hafta içinde düzelir. Nadiren, semptomlar 6 aydan daha uzun sürebilir. Antiinflamatuar ilaçların kesilmesi sonrası relapslar ortaya çıkabilir. Relaps hastaların yaklaşık olarak %10-15'inde ortaya çıkmaktadır. Rekürrenslerin çoğu operasyondan sonraki 6 ay içinde ortaya çıkmaktadır [67].

#### Morbidite / mortalite

DS genellikle hafif, kendi kendini sınırlayan inflamatuvar bir hastalık olarak kendini gösterir. Progresif perikardiyal efüzyona bağlı olarak hayatı tehdit eden perikard tamponadı gelişebilir. Tamponad DS'li olguların %1'inden daha az görülür. Kardiyak dolum basınçlarının artması, ventriküler diyastolik dolumun progresif kısıtlanması, kardiyak out put ve kalp debisinde azalmaya bağlı olarak kardiyak tamponad gelişir [68].

#### Caydırıcılık / Önleme

Günümüzde DS gelişimini önlemede kullanılan bir medikal ajan bulunmamaktadır [69]. Kardiyopulmoner bypass uygulanan hastalarda DS'yi önlemek için kortikosteroid ve kolşisin kullanımının koruyuculuğu gözlenmemiştir [70].

#### Çıkar Çakışması ve Finansman Beyanı

Bu çalışmada çıkar çakışması ve finansman destek alındığı beyan edilmemiştir.

#### Kaynaklar

- Dressler W. A post-myocardial infarction syndrome: preliminary report of a complication resembling idiopathic, recurrent, benign pericarditis. JAMA 1956; 160:1379-83.
- 2. Davidson CO, Oliver MF, Robertson RF. Post-myocardial infarction syndrome. BMJ 1961; 2:535-9.
- 3. Jaworska-Wilczynska M, Abramczuk E, Hryniewiecki T. Postcardiac injury syndrome. Med Sci Monit. 2011: 17(11):13-4.
- 4. Wessman DE, Stafford CM. The postcardiac injury syndrome: case report and review of the literature. South Med J 2006; 99: 206-7.
- 5. Soloff LA: Pericardial cellular response during the post-myocardial infarction syndrome. Am Heart J 1971;82(6):812-6.
- 6. Ito T, Engle MA, Goldberg Hp. Post-pericardiotomy syndrome following surgery for non-rheumatic heart disease. Circulation 1958; 17:549-56.
- 7. McGuiness JB, Taussig HB. The postpericardiotomy syndrome: Its relationship to ambulation in the presence of "benign" pericardial and pleural reactions. Circulation 1962; 26:500-7.
- 8. Henry SC, Hemngton RT, Wilcox BR: Postpericardiotomy syndrome with persistent pericardial effusion. South Med J. 1982; 75:597-8.
- 9. Engle MA, Zabriskie JB, Sentarfit LB, Ebert PA. Post pericardiotomy syndrome: A new look at an old condition. Mod Concepts Cardiovasc Dis 1975; 44:59-64.
- 10. Dressler W. The post myocardial infarction syndrome: A report of forty four cases. Arch Intern Med1959; 103:28-42.
- 11. Light RW. Pleural Diseases. 3rd ed. Baltimore; Williams & Wilkins, 1995: 225-9.
- 12. Miller RH, Horneffer PJ, Gardner TJ, Rykiel MF, Pearson TA. The epidemiology of the postpericardiotomy syndrome. Am Heart J 1988; 116:1323-9
- 13. Lichstein E, Arsura E, Hollander G. Greengart A, Sanders M. Current incidence of postmyocardial infarction (Dressler's) syndrome. Am J Cardiol 1982; 50(6), 1269-71.
- 14. Welin L, Vedin A, Wilhelmsson C. Characteristics, prevalence, and prognosis of postmyocardial infarction syndrome. Br Heart J 1983; 50:140-5.
- 15. Yan V, Hunt D, Sloman G. Pericardial pain in acute myocardial infarction. Eur J Curdiol 1973; 1:79-82.
- 16. Toole JC. Silverman ME. Pericarditis of acute myocardial infarction. Chest 1975; 67(6), 647-53.
- 17. Khan AH. Postmyocardial infarction syndrome. Unusual mode of presentation. Cardiology 1978; 63:188-92.
- 18. Janton OH, Glover R, O'Neil TJ, et al. Results of the surgical treatment for mitral stenosis: analysis of 100 consecutive cases. Circulation 1952; 6:321-33.
- 19. Burch GE, Colcolough HL. Postcardiotomy and postinfarction syndromes: a theory. Am Heart J 1970; 80:290-1.
- 20. Engle MA, Zabriskie JB, Senterfit LB, Gay WA Jr, O'Loughlin JE Jr, Ehlers KH. Viral illness and the postpericardiotomy syndrome: a prospective study in children. Circulation 1980;62:1151-8.
- 21. Khan AH. The postcardiac injury syndromes. Clin Cardiol. 1992; 15:67-72.
- 22. De Scheerder I, De Buyzere M, Robbrecht J, De Lange M, Delanghe J, Bogaert AM, et al. Postoperative immunological response against contractile proteins after coronary bypass surgery. Br Heart J. 1986;56(5):440-4.
- 23. Versey JM, Gabriel R. Soluble-complex formation after myocardial infarction. Lancet 1974; 2: 493-4.
- 24. Earis, JE, Marcuson, EC, Bernstein, A. Complement activation after myocardial infarction. Chest 1985; 87: 186-190.
- 25. Bendjelid K, Pugin J. Is Dressler Syndrome Dead? Chest. 2004; 126: 1680-2.
- 26. Cabalka AK, Rosenblatt HM, Towbin JA, Price JK, Windsor NT, Martin AB, et al. Postpericardiotomy syndrome in pediatric heart transplant recipients. Immunologic characteristics. Tex Heart Inst J. 1995;22(2):170-6.
- 27. Wessman DE, Stafford CM. The postcardiac injury syndrome: case report and review of the literature. South Med J. 2006; 99(3):309-14.

- 28. Nomura Y, Yoshinaga M, Haraguchi T, Oku S, Noda T, Miyata K, et al. Relationship between degree of injury at operation and the change in antimyosin antibody titer in the postpericardiotomy syndrome. Pediatr Cardiol 1994; 15: 116-120.
- 29. Sklaroff HJ. The post-pulmonary infarction syndrome. Am Heart J 1979; 98:722-6.
- 30. Engle MA. Postpericardiotomy and allied syndromes. In: Reddy PS(Ed). Pericardial Disease. Raven Press, New York 1982. 313.
- 31. Spodick DH. Infection and infarction: Acute viral (and other) infection in the onset, pathogenesis and mimicry of acute myocardial infarction. Am JMed 1986:81:661-8.
- 32. Light RW, Broaddus VC. Pleural Effusion. In: Murray JF, Nadel JA (Ed). Textbook of Respiratory Medicine. 3rd ed. Philadelphia: WB Saunders Company, 2000; 2032.
- 33. Ceylan E, Kanbay A, Günak F, Sezen Y, Eren TŞ. Dressler Syndrome is characterized by Massive Hemoptysis. J Clin Anal Med 2012; DOI: 10.4328/JCAM.1439.
- 34. Kırkıl G, Muz MH, Deveci F, Turgut T, Erdem E. Dressler Sendromu; Bir Olgu Nedeniyle. Fırat Tıp Dergisi 2008;13(3): 213-216.
- 35. Bajaj BPS, Evans KE, Thomas P. Postpericardiotomy syndrome following temporary and permanent transvenous pacing. Postgrad Med J 1999; 75: 357-8
- 36. Kelly BM, Nicholas JJ, Chhablani R, Kavinsky CJ. The postpericardiotomy syndrome as a cause of pleurisy in rehabilitation patients. Arch Phys Med Rehabil 2000:81:517-8.
- 37. Stelzner TJ, King TE, Antony VB. The pleuropulmonary manifestations of the postcardiac injury syndrome. Chest 1983; 84: 383-7.
- 38. Ofori-Kraykye SK, Tyberg TI, Geha AS, Hammond GL, Cohen LS, Langou RA. Late cardiac tamponade after open heart surgery: Incidence, role of anticoagulants in its pathogenesis and its relation to postcardiotomy syndrome. Circulation1982; 63:1323-8.
- 39. Ikaheimo MJ, Huikuri HV, Airaksinen KEJ, Korhonen U-R, Linnaluoto MK, Tarkka MR, et al. Pericardial effusion after cardiac surgery: Incidence, relation to the type of surgery, antithrombotic therapy, and early coronary bypass graft patency. Am Heart J 1988;116:97-102.
- 40. Claurec M, Cristol R, Raharissan S: Dressler's syndrome. Presse Medica-le.1966;74:965-8.
- 41. Prewitt JA: Cardiac tamponade as a late complication of open heart surgery. Am Heart J 1968;76:139-41.
- 42. Lawrence MS,Wright R: Tamponade in Dressler's syndrome with immunological studies. Br Med I 1972:1:655-6.
- 43. Arisoy E, Korkmaz İ, Eren ŞH, Güven FMK, Yilmaz MB, Beydilli İ, et al. Analyses of the Risc Factors for Atrial Fibrillation Among the Patients Who were Admitted to Emergency Service. J Clin Anal Med 2012;3(2):143-6.
- 44. Reinmüller R, Gürgan M, Erdmann E, Kemkes BM, Kreutzer E, Weinhold C. CT and MR evaluation of pericardial constriction: a new diagnostic and therapeutic concept. J Thorac Imaging. 1993;8(2):108-21.
- 45. Urschel HC Jr, Razzuk MA, Gardner M: Coronary artery bypass occlusion secondary to postcardiotomy syndrome. Ann Thorac Surg 1976; 22:528-31
- 46. Kossowsky WA, Lyon AF, Spain DM. Reappraisal of the postmyocardial infarction Dressler's syndrome. Am Heart J 1981;102:954-6.
- 47. Yıldızeli B, İsbir S, Dal D. Pulmoner Tromboendarterektomi. J Clin Anal Med 2012: DOI: 10.4328/ICAM.464.
- 48. Winterbauer RH. Nonmalignant Pleural Effusions. In: Fishman AP (Ed). Fishman's Pulmonary Diseases and Disorders. 3 nd eds.New York, McGraw-Hill 1998:1:89:1422-6.
- 49. Shahar A, Hod H, Barabash GM, Kaplinsky E, Motro M. Disapperance of a syndrome: Dressler's syndrome in the area of thrombolysis. Cardiology 1994; 85: 255-8.
- 50. Bartels C, Honig R, Burger G, Diehl V, de Vivie R. The significance of anticardiolipin antibodies and anti-heart muscle antibodies for the diagnosis of postpericardiotomy syndrome. Eur Heart I 1994:15:1494-9.
- 51. Engle MA, McCabe JC, Ebert PA, Zabriskie J. The postpericardiotomy syndrome and antiheart antibodies. Circulation 1974;49:401-6.
- 52. Engle MA, Gay WA, McCabe J, Johnson D, Senterfit LB, Zabriskie JB. Postpericardiotomy syndrome in adults: incidence, autoimmunity and virology. Circulation 1981;64:1158-60.
- 53. Webber SA, Wilson NJ, Fung MY, Malleson PN, Petty RE, Patterson MW, et al. Autoantibody production after cardiopulmonary bypass with special reference to postpericardiotomy syndrome. J Pediatr 1992;121:744-7.
- 54. Beynel P, Perol M, Nesme P, Guerin JC. Post-cardiac injury syndrome: an rare cause of pleurisy, value of pleural fluid analysis. Rev Mal Respir 2000; 17:1103-4. 55. Shrivastava R, Venkatesh S, Pavlovich BB, Bharadwaj J, Vaz A. Immunological analysis of pleural fluid in post-cardiac injury syndrome. Postgrad Med J. 2002;78:362-3.
- 56. Kim S, Sahn SA. Postcardiac injury syndrome. An immunologic pleural fluid analysis. Chest 1996;109:507-12.
- 57. Hajduczok ZD, Ferguson DW. Percutaneous balloon pericardiostomy for nonsurgical management of recurrent pericardial tamponade: a case report. Intensive Care Med. 1991; 17(5):299-301
- 58. Tapson VF. Acute pulmonary embolism. N Engl J Med 2008;358:1037-52.
- 59. Weiser NJ, Kantor M, Russel HK, Murphy L. Postmyocardial infarction syndrome. Circulation 1962;25,643-50.
- 60. Gregoratos G. Pericardial involvement in acute myocardial infarction. Cardiol Clin. 1996;8:601-18.
- 61. Hearne C, Forjuoh SN. Postcardiac injury syndrome after coronary angioplasty and stenting. J Am Board Fam Pract. 2003;16:73-4.

- 62. Horneffer PJ, Miller RH, Pearson TA, Rykiel MF, Reitz BA, Gardner TJ. The effective treatment of postpericardiotomy syndrome after cardiac operations. A randomized placebocontrolled trial. J Thorac Cardiovasc Surg 1990;100:292-6.
- 63. Little AG, Kremser PC, Wade JL, Levett JM, DeMeester TR, Skinner DB. Operation for diagnosis and treatment of pericardial effusions. Surgery. 1984;96(4):738-
- 64. Naunheim KS, Kesler KA, Fiore AC, Turrentine M, Hammell LM, Brown JW. Pericardial drainage: subxiphoid vs. transthoracic approach. Eur J Cardiothorac Surg. 1991:5(2):99-103
- 65. Madsen SM, Jakobsen TJ. Colchicine treatment of recurrent steroid-dependent pericarditis in a patient with post-myocardialinfarction syndrome (Dressler's syndrome). Ugeskr Laeger 1992;154:3427-8.
- 66. Forbes TJ, Horenstein MS, Vincent JA. Balloon pericardiotomy for recurrent pericardial effusions following fontan revision. Pediatr Cardiol. 2001;22(6):527-9.
- 67. Imazio M, Cecchi E, Demichelis B, Ierna S, Demarie D, Ghisio A, et al. Indicators of poor prognosis of acute pericarditis. Circulation 2007;115:2739-44.
- 68. Dorfman TA, Agel R. Regional pericarditis: a review of the pericardial manifestations of acute myocardial infarction. Clin Cardiol 2009;32:115-20.
- 69. Imazio M, Brucato A, Markel G, Cemin R, Trinchero R, Spodick DH, et al. Metaanalysis of randomized trials focusing on prevention of the postpericardiotomy syndrome. Am J Cardiol. 2011;108(4):575-9.
- 70. Finkelstein Y, Shemesh J, Mahlab K, Abramov D, Bar-El Y, Sagie A. Colchicine for the prevention of postpericardiotomy syndrome. Herz. 2002;27(8):791-4.

#### How to cite this article:

Ceylan E. Dressler Syndrome. J Clin Anal Med 2015;6(suppl 5): 718-23.

## A Case of Metachronous Lung Carcinoma



## Metakron Akciğer Karsinomlu Olgu

Metakron Akciğer Karsinomlu Olgu / A Case of Metachronous Lung Carcinoma

Levent Özdemir<sup>1</sup>, Burcu Özdemir<sup>2</sup>

¹Göğüs Hastalıkları, Dörtyol Devlet Hastanesi, ²Göğüs Hastalıkları, İskenderun Devlet Hastalıkları, Hatay, Türkiye

Akciğer kanserli hastalarda primer kanserin tedavisi sonrası ortaya çıkan 2. ve 3. primer tümöre metakron akciğer tümörü denir. Metakron tümörlerde 1975'te Martini ve Melamed'in 1995'te de Antakli ve arkadaşlarının tanımladığı kriterler kullanılmaktadır. Bu kriterlere göre metakron tümörlerde farklı histolojik tip veya histolojik tip aynı ise hastalıksız geçen sürenin 2 yıldan fazla olması, hastalığın in situ düzeyde saptanması, ikinci kanserin değişik bir lob veya akciğerde olması veya ortak lenfatik drenaj yolunda karsinom olmaması, ekstrapulmoner metastaz bulunmaması esas alınmaktadır[1].

55 yaşında erkek hasta, 2012 yılında sağ akciğer T2N1M0 (squamoz hücreli karsinom) tanısı ile opere edildi. Sonrasında hastaya kemoterapi ve radyoterapi tedavisi uygulandı. 2 yıl düzenli takip sonrası kontrolleri bırakan hasta 2015 yılında sol akciğer T2N0M0 (Squamoz hücreli karsinom) tanısı ile postoperatif sintigrafik akciğer alanı hesabı sonrası (Tahmini postop FEV1:0.93 lt) segmentektomi uygulandı.



Resim 1. 2012 PET CT Sağ akciğer alt lob superiyor segmentte 5,5x5 cm kitle



Resim 2. 2015 PET CT Sol akciğer alt lob superiyor segmentte 3x4cm kitle

## Kaynaklar

1. Turhan K, Yoldaş B, Çağırıcı U, Çakan A. Senkron ve metakron akciğer kanserlerinde cerrahi tedavi sonuçları. Ege Journal of Medicine 2009;48(2):85-8.

DOI: 10.4328/JCAM.3894 Received: 19.09.2015 Accepted: 30.09.2015 Printed: 01.10.2015 Corresponding Author: Levent Özdemir, Göğüs Hastalıkları, Dörtyol Devlet Hastanesi, Hatay, Türkiye. GSM: +905333888984 E-Mail: Levent2408@mynet.com

# A Case of Hydatid Cyst with Localization in Anterior Mediastinum



## Anteriyor Mediastinal Yerleşimli Kist Hidatik Olgusu

Anteriyor Mediastinal Kist Hidatik / Anterior Mediastinum Hydatid Cyst

Gökhan Perincek<sup>1</sup>, Levent Özdemir<sup>2</sup>, Burcu Özdemir<sup>3</sup>

<sup>1</sup>Göğüs Hastalıkları, Kars Devlet Hastanesi, Kars, <sup>2</sup>Göğüs Hastalıkları, Dörtyol Devlet Hastanesi, Hatay, <sup>3</sup>Göğüs Hastalıkları, İskenderun Devlet Hastalıkları, Hatay, Türkiye

Hydatid cyst is an endemic public health problem in our country. While it often develops in lungs and liver, it may also affect all tissues. Extrapulmonary intrathoracic hydatid cyst is rarely seen as the involvement of thoracic wall, mediastinum, pericardium, myocardium, fissure and pleural cavity. Mediastinum involvement by hydatid cyst is quite rare (0.1%-0.5%) [1].

A 56-year-old male patient had complaints of chest pain and coughing. No specific features were detected in his personal medical history. His chest radiograph revealed an evenly bordered opacity in the left hilar region and bronchiectatic areas were in left lower zone (Image 1) and his thoracic tomography revealed a 5.5 x 4.5 cm hydatid cystic lesion of which wall had calcifications and lumen contained collapsed membranes (Image2-5). Mediastinal MR imaging of the identified lesion supported the finding of a hydatid cyst as well (Image6-7). Abdominal ultrasound was normal. The patient underwent surgery. The case was reported as hydatid cyst.



Image 1. Chest radiograph opacity in the left hilar region

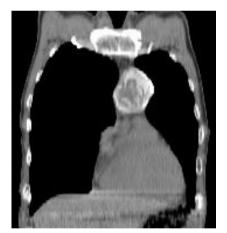


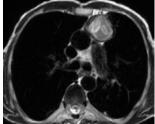
Image 2. 5. Thoracic tomograph 5.5 x 4.5 cm hydatid cystic lesion

Image 6. 7. Mediastinal MR hydatid cys









#### Reference

Image 4.

1. Özyurtkan MO, Koçyiğit S, Çakmak M,Özsoy İE, Balcı AE. Case Report: Mediastinal Hydatid Cysts. Türkiye Parazitol Derg 2009; 33:179 -181.

DOI: 10.4328/JCAM.4237 Received: 20.12.2015 Accepted: 03.01.2016 Printed: 01.10.2015 Corresponding Author: Levent Özdemir, Göğüs Hastalıkları, Dörtyol Devlet Hastanesi, Hatay, Türkiye. GSM: +905333888984 E-Mail:Levent2408@mynet.com





# Uzun Atımlı Nd-YAG Lazerle Tedavi Edilen Telanjiektatik Rozase Olgusu

Nd-YAG Lazerle Tedavi Edilen Rozasea / Rosacea Treated with Nd-YAG Laser

Can Ergin Dermatoloji Kliniği, Dışkapı Yıldırım Beyazıt Eğitim Araştırma Hastanesi, Ankara, Türkiye

#### Editör için:

68 yaşında erkek hasta, yüzünde hafif eritem, bir kaç adet papül ve özellikle burun üzerinde yoğunlaşan telanjiektazilerle kliniğimize başvurdu. Hastanın öyküsünde rozase tanısıyla yıllardır ultraviyole koruyucu kremler, topikal metranidazol ve zaman zaman oral doksisiklin 100mg/gün kullandığı öğrenildi. Bu tedavilerle yüzdeki eritem, papül ve püstüller gerilemiş, ancak telanjiektaziler yıllar içersinde artmıştı(Resim1). Hastanın burun üzerindeki telanjiektazilerine bir seans uzun atımlı Nd-YAG lazer (Fotona XP-2 Focus 2013, Slovenya) uygulaması yapıldı. Lazer parametreleri; ışın çapı 2mm, atım süresi 15 ms, fluens 160j/cm2 ve frekans 1,5Hz idi. Bu parametrelerle telanjiektaziler 2 kez tarandı. Hastanın burun üzerinde 5 yıldır mevcut olan telanjiektazilerinde tama yakın düzelme gözlendi(Resim 1). Bir ay sonraki konrol muayenesinde burun üzerinde çok az miktarda ince telanjiektazilerin kaldığı görüldü. Bununla birlikte, hasta kozmetik olarak tedavi sonucundan memnun olduğu için, tekrar lazer uygulaması istemedi. 6 aylık takipte tedaviye bağlı skar veya başka bir yan etki izlenmedi.

Rozase, yüzde eritem, telanjiektazi, papül ve/veya püstüllerle karakterize kronik enflamatuar bir deri hastalığıdır. Hastalık her iki cinsi de etkilemekle birlikte, 30-50 yaş arasında açık tenli kadınlar daha sık etkilenir. Hastalığın etyopatogenezinde genetik yatkınlık, enfeksiyöz ajanlar, çevresel faktörler, güneş ışığı maruziyeti, psikolojik stres ve vasküler hiperreaktivite gibi etkenler suclanmıştır[1].

Rozase tedavisinde başlıca kullanılan topikal ajanlar; brimonidine tartrate, pimekrolimus, takrolimus, metronidazol ve azeleik asittir. Sistemik tedavide oral tetrasiklinler veya isotretinoin tercih edilir[2]. Vasküler lazerler ise, rozasede eritem ve telanjiektazilerde etkili bulunmuştur. En sık kullanılan lazerler, pulsed dye lazer, potassium-titanyl-phosphate(KTP) ve neodymium-yttrium aluminum garnet(Nd-YAG) lazerlerdir. Nd-YAG lazerler, damar hemoglobini hedefleyip fotokoagülasyona neden olarak damarları yok ettiği düşünülmektedir[3].

Literatürde, Nd-YAG lazerle rozase tedavisi bildiren yayın sayısı çok azdır. Salem ve ark. 15 rozase hastasının yüzünün bir tarafına Nd-YAG lazer (ışın çapı: 18mm, atım süresi: 10 ms, fluens: 22j/cm2) diğer yüzlerine PDL olmak üzere, 4 hafta arayla

toplam 3 seans tedavi uygulamışlar. Nd-YAG lazeri PDL'den daha etkili bulmuşlardır[4]. 16 rozase hastasını içeren diğer bir çalışmada, hem Nd-YAG lazer(ışın çapı: 8mm, atım süresi: 0.3 ms, fluens: 6j/cm2) hem de PDL rozase eriteminde etkili bulunmuştur[5]. Bizim hastamızda ise Fotona XP-2 Focus lazer ile, ışın çapı: 2mm, atım süresi:10 ms, fluens:160j/cm2 ve frekans 1,5Hz parametreleri kullanılarak, yaklaşık 5 dakika süren bir seans sonrası hastanın burun üzerindeki telanjiektazilerinin tama yakın kaybolduğu görüldü.

Sonuç olarak, Nd-YAG lazer uygulamalarının ileride rozase hastalarındaki özellikle eritem ve telenjiyektazilerin tedavisinde güvenli ve etkili bir seçenek olacağını düşünmekteyiz.



Resim 1. Hastanın tedavi öncesi(sol), ve tedavi sonrası görüntüleri(sağ)

#### Kaynaklar

- 1. Ekiz Ö, Balta İ. Acne Vulgaris and Acne Rosacea: An Update in Etiopathogenesis. J Clin Anal Med 2014; DOI:10.4328/JCAM.2423.
- 2. Weinkle AP, Doktor V, Emer J. Update on the management of rosacea. Clin Cosmet Investig Dermatol. 2015; 8: 159-77.
- 3. Mazer JM. Role of laser in the treatment of rosacea. Ann Dermatol Venereol 2014;141:175-8.
- 4. Salem SA, Abdel Fattah NS, Tantawy SM, El-Badawy NM, Abd El-Aziz YA. Neodymium-yttrium aluminum garnet laser versus pulsed dye laser in erythemato-telangiectatic rosacea: comparison of clinical efficacy and effect on cutaneous substance (P) expression. J Cosmet Dermatol 2013;12(3):187-94.
- 5. Alam M, Voravutinon N, Warycha M, Whiting D, Nodzenski M, Yoo S, et al. Comparative effectiveness of nonpurpuragenic 595-nm pulsed dye laser and microsecond 1064-nm neodymium:yttrium-aluminum-garnet laser for treatment of diffuse facial erythema: A double-blind randomized controlled trial. J Am Acad Dermatol 2013;69(3):438-43.

DOI: 10.4328/JCAM.3819 Received: 07.08.2015 Accepted: 31.08.2015 Printed: 01.10.2015 Corresponding Author: Can Ergin, Dermatoloji Kliniği, Dışkapı Yıldırım Beyazıt Eğitim Araştırma Hastanesi, Kavaklıdere, Ankara, Türkiye. GSM: +905056831264 E-Mail: drcanergin@hotmail.com

**Author Information:** Following guidelines were prepared for providing unity in the format of the articles submitted to our journal. Please send your articles in accordance with these rules.

Journal of Clinical and Analytical Medicine is international openaccess journal and peer-reviewed scientific articles in every branch of medicine concerned with the retrospective, prospective or experimental studies. Our Journal is dedicated to disseminate unique papers for free to all researchers, scientists and clinicians to promote and share progressions within the scope of the journal. The journal is published six times in a year and in January, March, May, July, September and November. The author(s) undertake(s) all scientific responsibility for the manuscript. All the printed and digital content of this journal is archived in the ULAKBİM National Databases.

**On-line Mailing Address:** Correspondence and article submissions are made at the www.jcam.com.tr address by on-line. For each article sent, it will be confirmed that the manuscript was received via e-mail.

**Manuscript language:** Articles in English are accepted. Articles and abstracts should be assessed by language expert before being sent. In addition, approval should be specified in the presentation to editor page. If the language expert of the article is not one of the authors, the person's name must be implied in the Acknowledgement section at the end of the article. Spelling and grammar errors in the article sent to the editorial board can be corrected without touching the core of the article.

A statement of the manuscript is not published elsewhere: Each author should state that the submitted article partly or completely has not been published elsewhere previously or is already in assessment in printed form or as digital media. This rule is true for symposium, information transmission, books, articles written by invitation, submissions in electronic format, and all sorts of prenotification (except for up to 400 words abstracts). If there is quotation from the previously published articles, the authors must take written consent of the author of the article and of the copyright holder and the consent must be specified in this article. The consent document, together with the article should be sent to our editor. Studies previously presented in congress as orally or poster must be specified in the title page together with date, conference name and location.

Assessment: The format of the article submitted to journal is checked firstly. Articles that do not comply with the format will be returned to the author without further review. Therefore, to avoid loss of time and labor, authors should review the rules of the journal carefully. For each article reviewed for publication, two or more referees will be assigned domestic or from abroad (The referee can be assigned from outside the council). Manuscripts are evaluated by taking into account the importance, originality, and scientific validity. This review of the articles will be evaluated again by the editorial board. The editorial board can make necessary arrangements without changing the contents of the text of the accepted articles to journal. Journal has the authority to do corrections in writing to, request changes in the format, and shortening with informing the authors. Regarding articles will not be included in the publication program until the desired edits are done. None of the authors' name can be deleted from the list without the written consent of all authors after manuscript has been sent to journal for publication. Similarly, no names can be added as author and the author order can't be changed.

**Acceptance for publication:** Submitted manuscripts will be ordered for publication with the approval of the editorial board. Unaccepted manuscripts and attachments (photos, tables, figures,

CD, disk...etc.) are not sent back unless otherwise specified.

The assignment of copyright: Authors must transfer copyright to the Clinical and Analytical Medical Journal. According to the 1976 Copyright Act, any publication rights of the articles accepted for publication belong to publisher institutions. This assignment becomes binding with accepting of the article for publication. Content of the articles is entirely the responsibility of the author. Article authors will not be paid any fees for articles. Any part of the printed material can't be used elsewhere without the written permission of the Clinical and Analytical Medical Journal. The authors should fill the "Copyright Transfer Form" presented in the attachment and submit to the www.jcam.com.tr address as on- line with the article.

**Rules for following while writing manuscripts:** Manuscripts and all related figures, tables and photos should be sent as on-line via the Internet.

**Checklist:** The following list is presented to authors for checking manuscripts and completion of deficiencies before sending to journal. Incomplete articles will not be evaluated.

General: Manuscripts should be written using preferably Microsoft Word program. Manuscripts should be written using 12 point Arial or Times New Roman characters. Writings should be written on one side of A4 (21x29.7 cm) white paper file, double spaced throughout the article (Title, Turkish and English abstracts, article, references, tables and also subtitles) and 3 cm from all edges of the page. The first author's last name, including the title page must be located in the upper right corner of every page. Manuscripts should be prepared in the following order: (1) Turkish title, abstract and key words, (2) English title, abstract and key words, (3) article, (4) acknowledgement (if any), (5) funds and organizations supporting (if required to declare), (6) references (7) tables and / or figures and (8) subtitles of figures. All pages are numbered in sequence from the bottom right corner given the number (1) after the Turkish title page.

**Original research and review manuscripts:** Manuscripts should not exceed twenty pages totally; 1 for Turkish (title abstract, key words) and 1 for English (title abstract, key words), 10 pages for article (maximum 2500 words), 4 pages for tables and / or figures (including subtitles page), 3 pages for references.

**Review manuscripts:** Review articles are prepared directly or by invited authors. For all kinds of medical issue, articles should be prepared including current medical literature. 4000 words should be limited to the reviews of the manuscripts.

Case reports and surgical technique manuscripts: Manuscripts should not exceed 8 pages (1000 words) excluding title page, Turkish and English abstracts, and references. Abstracts should be 100-150 words without diving to parts (English and Turkish). Surgical technique manuscripts are the articles describing detailed operation techniques. These manuscripts should include a detailed visual description.

Letters to the editor: Letters are replied by editor or the author(s) of the article in published form. It includes different opinions, experiences, questions and objections of the readers regarding the articles published in journals in the last year. There are no title and summary sections. It should not exceed two pages totally (500 words maximum) and number of references must be at most 4. Tables and figures can be printed only in exceptional cases. The number and date of the dedicated article should be specified and in the end the author's name, affiliation, address must be provided.

**Editorial Comments/Discussion:** It includes comment by the experts except the author of research that related to published original research articles. It is published at the end of the article related to the subject. Editorial manuscripts should be limited to 2500 words.

**Medical Education:** They are scientific articles of current medical information to readers also supported by the current clinical and laboratory applications. They consist of abstract, headlines of the regarding issue, and references sections. Scientific Letter: It is manuscript that provides general information on medical issues to the reader and discusses the subject by referring to scientific articles. It consists of the abstract, headlines of the regarding issue, and references sections.

**Original Images:** They are medical images with explanations seen rarely in the literature. They consist of a summary related to the subjects, and the original figures.

**For video (only on-line review):** Avi, mov, mpg or rm files should be preferred (features; resolution: 320 x 240 pixel, time: maximum 30 seconds, film frame/second rate: 20-30). In still images, jpg (inch/72 dot.) format should be preferred. It should be installed as supplemental file.

Title page: Titles that are clear and as short as possible are preferred. Title of the article should not exceed 100 characters (each letter and space is considered 1 character) in the research and review articles but in the case reports, letters and surgical technique manuscript it should not exceed 85 characters. Title should be written in Turkish and English. Just below the title, a short title (running title) should be added not exceeding 40 characters. Do not use abbreviations in the title. Name and surnames of the authors only who contribute to work directly must be written clearly, and the number of the authors should not exceed 8 in the research articles, 5 in the case presentations and surgical technical articles, and 3 in the letters to editor. If the number of authors exceeds the limits, the reason should be explained to editor. Name of the study institution and city of institution should be written below the authors' name. If more than one institution has participated in the study, the institution must be specified for each author (Authors should not use their titles in any place of the manuscript). Funds and supporting organizations to work should be indicated in the title page. The conference's name, date (day/month/year) and place of the studies which are planned to be presented in any congress or was previously available as a poster or oral presentations should be specified to the editor. The author's contact information should be written on the bottom of the title page for contact (Author's name, last name, exact address, postal code, telephone number, fax number and especially e-mail address should be written).

Abstracts: Abstracts must be written in Turkish and English, should be limited to 200-250 words for research papers. In abstracts, title should not be used (English title should be written on the title page). Turkish and English abstracts should consist of four parts (Introduction, Material and Methods, Results, Discussion). For case reports, reviews and surgical techniques these sections are not required and abstracts should not exceed 100-150 words. In the medical education, and scientific letters, the abstracts should be limited to 100-150 words and the sections are not required. Letters to the editor, commentaries, editorial articles and original images do not require summary. In the abstracts, use of abbreviations should be avoided as possible. If it is absolutely necessary to use abbreviation, it should be used where it first passes in parentheses after being defined.

Key Words: At least 2 Turkish and English key words should be giv-

en. Words should be separated from each other by semicolon (;). English key words should be taken from www.nlm.nih.gov/ mesh/ MBrowser.html and Turkish key words should be taken from www. bilimterimleri.com (Medical Subject Headings and Türkiye Bilim Terimleri). They should be given after abstracts.

Text: Text should consist of Introduction, Materials and Methods, Results and Discussion section. Introduction section, should explain the issue and the purpose of study. In the Materials and Methods section, the place of work, time, and study plan must be stated. If there are subtitles, characteristics of cases, surgical techniques or methods of experimental studies, data collection and follow-up method, and statistical methods (detailed) used should be stated in the form orderly as stated here. In results section, the data obtained, together with the statistical results should be given. In discussion section, the study results are evaluated by comparison with the other papers and especially with national ones. References, figures and tables should be numbered in order of the transition within the article. Abbreviations should be avoided as possible; if the abbreviation is used, the expression should be at the location of the first pass. In Turkish papers, Turkish expression and abbreviations should be used as possible. All measurement units in the text should be given in accordance with international metric standards. Footnotes should be given at the bottom of the mention page. For medicine, equipment and other materials in paper, the company name and location must be specified in parentheses. The acknowledgement including the grants and financial support or technical assistance should be at the end of the article before the reference section.

References: Authors must be selective in the use of references and references directly relevant with the study should be included. Number of references should not be more than 25 in research articles, 8 in case presentations, and surgical technical articles, 85 in reviews, and 4 in letters to the editor. Reference number should be given at the end of the sentence in brackets "[]" before point mark, if name will not be given within the sentence. Exchange of information during personal contact and personal experience, manuscript during preparation and other unpublished data should not be given as the reference. References should be written to a separate page as double spaced, and numbered in order of transition in the text. Preference in the references should be given to local literature in Turkish and English texts. In journal references, year, volume, start and end pages are given; but in book references, the only year, start and end pages must be specified. These rules should be followed if references are shown in text; If the first author's last name will be given for the article's reference, "et al." should be added afterwards and the reference number should be placed in "[]" just after it and the sentence should be completed subsequently. If name will not be given within the sentence, the reference number should be given at the end of the sentence before point marks in the brackets "[]". If different references to show different expressions will be given in the sentence, they should be given before the punctuation mark in the brackets "[]" at the end of each reference relevant. If there are more than two consecutive references, the first and last ones, between "-" sign should be provided as [2-6]; [11-17]; [9-13]. Surname and first letters of the first name of the authors should be written in references. All authors must be specified if the number of authors is less than or equal to 6 and, if the number of authors is higher than 6, "et al." should be added at the end of the first 6 authors (for international papers) or "ve ark." (for national papers). Names of journals should be abbreviated according to Index Medicus. Authors are responsible for the reference and the accuracy of spelling. References should be written according to the places in the article, and specified with the sign of "[]" in text the end of sentence just after punctuation. Congress presentations, personal experiences, unpublished manuscripts, thesis and internet sites are not accepted as references. 'DOI' is the only acceptable on-line reference. Spelling and punctuation of the references should be suitable to examples listed below.

Examples for references (Pay attention to punctuation marks): For international published articles: Çubuk EC, Karakurt T. Surgical management of chest wall tuberculosis. J Cutan Med Surg 2009;13(1):33-9.

For national references: Ekici HA, Yucel B, Savaş MN. Proantosiyanıdın nitrojen mustarda bağlı akciğer hasarını azaltır. Gülhane Tıp Derg 2008;50(4):267-72.

**For journal supplements:** Yildırım SB. The cost of schizophrenia. J Psychiatry 1999;49(Suppl.3):S85-9.

For books: Savaş Z, Batuhan M, editors. Vascular and endovascular surgery. London: W. B. Saunders; 1999. p. 87-93

**For a chapter from the book:** Tosun R, Eken A, Savaş MN. Hypertension and stroke. In: Laragh JH, Brenner BM, editors. Hypertension: pathophysiology and management. 2nd ed. London: Ran Press; 1985. p.495-8.

**Tables:** Tables should be written on a separate page as two lines spaced. At the top of each table number and descriptive information should be specified. If there are abbreviations used in the table, the expansions of abbreviations should be written in the form of subtitles and alphabetical order. If previously published tables as a printed or electronic form used, written permission from the author and the publisher should be obtained. This written permission should be sent by fax or mail the journal editor. Transverse and longitudinal lines should not be drawn in the tables; just a straight line should be drawn top and bottom. Tables should not be submitted as such placed in the manuscript. Every table should be on a separate page and in the file of article sent.

Figure, Graphics, Pictures, and Subtitles: Subtitles should be written as double spaced on separate page. These should be numbered in accordance with their order within the text. If abbreviations are used n figures, graphics and pictures, expansions of abbreviations in alphabetical order below as subtitles should be included. Articles with tables, figures, and graphics embedded in the manuscript should not be sent. In microscopic images, the magnification ratio and staining technique should be described. When benefited from previously published figure, graphics, and pictures as printed or electronic form, written permission from the author as well as printing office should be taken. This written permission should be sent by fax or mail editor of the journal. Written permission should be taken from cases, if their images are used as recognizable. Descriptions of figures of the article should be written at the end of the file sent. Where tables, figures and graphics pass in the text should be specified. Images/photos should be colorful, and details should be clear in visible contrast. Figures and images/photos should be added to the system as separate jpg files. Image and photo files should be 100 pixel/inch, 8 cm width, and must not be less than 300 dpi resolution.

**Originals of the Drawings:** After articles accepted for publication, the journal editor may want the authors to send the originals of the images, drawings, and pictures sent in electronic media if they are not suitable for printing. Requests in this matter will be reported by the journal editor.

**Ethical Responsibilities:** Human Research: Ethical principles followed by researchers with the approval given by the ethics board and human research committee in the center of the study must be specified in the Materials and Methods section. Also statement of informed approval about it from every participant must be included. In such studies, the authors must specify that they comply with

the international guidelines (Declaration of Helsinki), the "Regulations on Pharmaceutical Research" enforced by The Ministry of Health of Turkey published in the 27089 numbered Official Journal dated 23 December 2008, and the other regulations published later. Authors should send a photocopy of the official seal of Ethics Board approval from the institution. If Photo ID is used to identify the patient, the patient's written consent must be submitted.

**Experimental Animal Studies:** Experimental Animal Studies: In Materials and Methods section, it should be expressed that in the experimental studies that all animals are subject to the ethical treatment in accordance with Guide for the Care and Use of Laboratory Animals. Studies on animals require ethical approval. A photocopy of the official seal of ethical approval must be submitted to our editor. Also, in the paper, the method of the analgesia and sacrifice applied to subjects should be written in a clear manner.

**Conflict of Interest:** Journal of Clinical and Analytical Medicine requests the authors to state all the financial resources that may be potential conflict of interest regarding their manuscript. When editors detect any potential upcoming conflicts, they contact the authors to remedy any discrepancies. We publish declaration of authors whether or not conflict of interest exists.

Scientific Responsibility Statement: Authors should declare that they are responsible for the article's scientific content in the Copyright Transfer Form of submitted article. These responsibility areas include topics such as study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation, and scientific review of the contents and approval of the final version of the article.

**Sponsorship Statement:** Authors if any must declare who do sponsorship and their roles in the study.



# **COPYRIGHT TRANSFER FORM**

Dear Editor.
--------------

Dear Editor,				
We send our article wi	th the wish of publishment:			
As we are the author(s) of the article named above, as a first author of my (our) manuscript, I (we) accept and state that the article; all kinds of copyright of the article are owned by Journal of Clinical and Analytical Medicine (Klinik ve Analitik Tip Dergisi); it is appropriate to the writing, publication, and ethics rules of the journal stated; it is an original work and was not published in Turkish or other languages in domestic or abroad previously or it is not under evaluation for publication; the scientific and ethical responsibility is to me (us); In case of other authors can not reach, all the authors are aware of the study. I undertake the other authors' responsibilities. The information in the article is accurate and real. May occur due to errors and deficiencies Journal of Clinical and Analytical Medicine (Klinik ve Analitik Tip Dergisi) is not a legal responsibility.				
Corrresponding Auth	nor:			
Name and surname	:			
Adress	:			
Phone	:			
Fax	:			
E-mail	:			
The author(s) name		Signature/Date		